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TEXAS STATE JOURNAL OF MEDICINE

Volume 47 Number 1 Austin, Texas, January, 1951

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1951 CURRENT THERAPY

This is the third in a series of annual volumes on today's best treatments. The 1949 and 1950 editions were used by tens of thousands of doctors, and now 1951 Current Therapy is ready. It follows the same successful basic plan of its predecessors, but 49 new contributors have been added and about 50% of the material is either brand new or extensively revised from last year. A very important feature is the addition of a *Roster of Drugs,* which lists alphabetically all drugs mentioned in the book and gives manufacturing and packaging data, dosages, solution percentages, etc. The subjects of Aviation Medicine, Parenteral Fluid Therapy in Children, and Cushing's Syndrome are completely new.

What you get, then, in 1951 Current Therapy is the *best treatment known to medical science today for every disease you're likely to encounter.*

1951 Current Therapy. An annual volume by 269 American Authorities selected by a special Board of 12 Editorial Consultants. Edited by HOWARD F. CONN, M. D. 700 pages, 8"x11". $10.00. *New—Ready February, 1951.*

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Texas State Journal of Medicine is published monthly by the State Medical Association of Texas. Entered as Second Class Matter July 1, 1905, at the Post Office at Fort Worth, Texas, under act of March 3, 1879. Postmaster send all notices on Form 3579 to 700 Guadalupe Street, Austin, Texas.

President's Page

PROLOGUE TO 1951

At the moment we are looking into 1951. The thoughts, the experiences, the deeds, the losses, the achievements of 1950 now are recorded in the pages of history.

Many of the pages in 1950 and in the preceding few years will be rather unpleasant reading in the future; they relate to some distressing facts. After 1945 the United States had hopes that the world had been made safe for democracy. Since then intense efforts have been made toward the development of a welfare-state planned economy. No group of citizens is more aware of this than doctors; yet they are by no means the only ones so impressed.

We realize that it is wise to have full and free discussion of all governmental philosophies, but it is not wise for certain ideologies to be forced too energetically by governmental agencies. In so doing a nation may lose sight of the necessity of protecting its people against other countries about whom so little apparently is known.

Thus, we find 1950 closing its pages with the dreadful story that again we are engaged in a war which we did not know was coming and for which we certainly were unprepared. We had been led to believe that peace was nearer than it had been for a long time. Truly 1950 has contained many pages of very dark and bitter history.

Interposed between these dark pages, however, will be found some bright ones which will stand out prominently before all ages in time to come. On them will be the records of heroes who have fallen in battle. The deeds of fighting men who could not ask why they were fighting will be recounted. We do not know what their hearts longed for in those trying hours, but from a military standpoint history probably will surmise that their thoughts were, "Too little given us too late." Now an everlasting obligation rests upon us to make and keep America great and free, as they would want it to be.

Other pages in the history of 1950 and the immediate past also will be bright. They will reflect that men and women in all walks of

life, in all professions, and in all industries have sensed political and governmental attitudes, goals, and objectives and even our plight. These persons recognize that freedom is something to be achieved only by great sacrifice; it is to be guarded and at all times defended at any cost regardless of the sacrifice. They dare to brave serious opposition in order to express their views to many people who have been led to believe that the security of mankind is to be found in federal control and direction. Surely this doctrine is not true, and the closing chapter of 1950 should warn, "People of the United States, wake up and think carefully about your government." In 1951 the voice of the people of the United States must be heard more loudly and far more frequently in Washington.

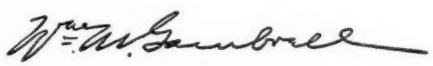
Doctors always have put the medical interests of the public first. Let us forget ourselves more than ever in 1951 and do a better job of caring for the medical interests of the public than ever before. We can care better for the health interests of our communities than Washington and we know it. It is our duty to prevent the public from being deceived and from being led to think otherwise.

Our pledge should be to do that. We should hope that Congress will devote itself less to consideration of a welfare-state planned economy. We should also hope that Congress will concentrate on the solution of those international problems which have been developing a number of years and which now threaten to destroy us. To this end we doctors must back Congress to our limits.

It is humanly impossible for any Congress to give due attention to the hundreds of welfare bills which are poured before it in times of peace. War imposes even greater burdens on our Congressmen, who are weighed down with the present international conflict.

Our final hope should be that the people will realize the seriousness of the tasks imposed upon Congress at this time. We must help them recognize the necessity of relieving Congress of the highly controversial and imprudent legislation with which it has been confronted while it attempts to take care of grave international problems.

Sincerely,



TEXAS STATE JOURNAL OF MEDICINE

DEVOTED TO THE INTERESTS OF THE
MEDICAL PROFESSION AND PUBLIC HEALTH IN TEXAS

Owned by the State Medical Association of Texas
and issued monthly by the Board of Trustees

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Editorial Offices, 700 Guadalupe Street, Austin, Texas
Publication Offices, 1114 West Daggett Street, Fort Worth 1, Texas

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Volume 47 **Austin, Texas, January, 1951** **Number 1**

HIGHLIGHTS OF CLEVELAND CLINICAL SESSION

The appropriation of a half million dollars as the nucleus of an annual fund to be raised for the aid of medical schools was one of the most important actions of the Board of Trustees of the American Medical Association which was taken at the Cleveland clinical session from December 5 to 8.

The action of the Board, which is expected to have far-reaching effects, received enthusiastic support from the House of Delegates. Within hours after the decision was announced, more than $6,000 had been pledged by individuals and several thousands more from other sources. Details have not been worked out yet; however, it is believed that a nonprofit corporation will be established to administer the fund and to solicit contributions. Those who wish to make an immediate contribution may obtain additional information from Dr. George F. Lull, Secretary of the Association.

The House of Delegates approved the Board's appointment of "a committee of appropriate size to study carefully the Twelve Point Program with the idea of making changes which may be indicated." The Board's recommendation that the committee be a continuing one to advise the Association concerning appropriate changes in policy which may be made advisable by altered circumstances was approved also. Chairman of the committee is Dr. Walter B. Martin, Norfolk, Va., and other members are Dr. Edwin S. Hamilton, Kankakee, Ill., and Dr. Leonard W. Larson, Bismarck, N. D.

The section of Board of Trustee's report concerning the Student American Medical Association was adopted by the House. Previously the deans of all medical schools in the United States had been written and the number of organized student bodies determined. A constitutional convention was authorized, and at a meeting December 28 and 29 at Association headquarters in Chicago, student delegates from 48 medical schools in the United States with a total enrollment of 15,855 organized the Student American Medical Association. Officers were elected as follows: Warren R. Mullen, Jackson, Mich., student at the University of Michigan Medical School, Ann Arbor, presi-

dent; Harry W. Sandberg, Moline, Ill., student at the University of Illinois College of Medicine, Chicago, vice-president; and David Buchanan, Huron, S. D., student at the University of South Dakota School of Medical Sciences, Vermillion, treasurer. Charles Wilson, Galveston, representing the University of Texas School of Medicine, was elected one of the seven members of the executive council. Leo Brown of Harrisburg, Pa., is the executive secretary and will make his headquarters at the American Medical Association in Chicago.

The student house of delegates has named its president and vice-president as representatives to the A.M.A. House of Delegates. A change in the constitution of the A.M.A. to provide such representation is expected to become effective at the annual meeting in Atlantic City in June.

A report on the results of the advertising campaign of the A.M.A. last October made at the clinical session revealed that in connection with the campaign, more than 65,000 persons in approximately forty businesses, professions, industries, and trades advertised their views on the issue of compulsory versus voluntary health insurance. Individual advertisers numbering 65,246 who took part in the campaign bought 1,186,594 inches of advertising space in addition to the newspaper advertising space sponsored by physicians in their nationwide schedule.

The scientific sessions, scientific and technical exhibits, color telecasts of surgical procedures and demonstrations, medical motion pictures, and House of Delegates meetinge were well attended; of 198 eligible delegates in the House of Delegates, 195 were seated on the opening day. Emphasized in the general topics of the scientific program were cancer, diseases of the heart and blood vessels, diabetes, diseases of children, surgery, anesthesia, and poliomyelitis. More than eighty physicians participated in lectures and demonstrations. Seventy exhibits, including practical laboratory demonstrations, were essentially devoted to the subjects mentioned, and more than 145 firms were represented in the technical exhibits. The several thousand visitors included physicians, medical students, nurses, exhibitors at the technical exhibits, and guests.

Texas again was well represented at the clinical session. The choice of the State Medical Association for General Practitioner of the Year, Dr. Jim Camp of Pecos, was one of three runners-up for the national honor; Dr. Dean Sherwood Luce, Canton, Mass., finally was chosen by the A.M.A. House of Delegates. Texans present in the House of Delegates were as follows: Drs. Truman C. Terrell, Fort Worth; J. B. Copeland, San Antonio; B. E. Pickett, Sr., Carrizo Springs; Robert B. Homan, Jr., El Paso; Allen T. Stewart, Lubbock; and John H. Wootters, Houston. Dr. F. J. L. Blasingame, Wharton, who serves as a trustee in both the State Medical and American Medical Associations, also attended as did Tod Bates, Executive Secretary, and Philip R. Overton, General Attorney, of the State Medical Association. Representing the scientific sessions of the A.M.A. were Dr. Charles T. Stone, Galveston, and Dr. Everett C. Fox, Dallas. Dr. Ernest E. Muirhead, Dallas, gave the paper "Acute Renal Failure: Causes, Differentiation and Treatment Following Incompatible Blood Transfusion; Experience with the Management of 32 Patients."

Another distinction for Texas was the announcement that Dr. J. T. Billups, Houston, had been appointed by the Board of Trustees to be chairman of the local committee on arrangements for the Houston clinical session, which will be held from December 4 to 7, 1951. Dr. Billups and Dr. Thomas J. Vanzant, Houston, attended the clinical session in Cleveland. Dr. R. W. Kimbro, Cleburne, chairman of the Committee on Public Relations, and Millard J. Heath, Dallas, Executive Secretary of Dallas County Medical Society, were among Texans who were present at the medical public relations conference immediately preceding the clinical session.

The 1951 annual meeting of the A.M.A. will be in Atlantic City from June 11 to 15. The 1952 clinical session will be held the first week in December in Denver and the 1953 clinical session in St. Louis at a date to be set later.

The furtherance of county medical society public relations was the object of the third annual national Medical Public Relations Conference held December 3 and 4 in Cleveland, just prior to the clinical session. More than 350 persons attended, including physician chairmen of state public relations committees, executive secretaries of county and state medical societies, trustees and other officers of the American Medical Association, and national officers of the Woman's Auxiliary. Representatives of numerous other national health organizations, publications, and radio systems also sat in on the two-day meeting.

Problems and strong points of medical public relations in both metropolitan and rural areas were emphasized at the conference. National, state, and county medical leaders and executives were enabled to share experiences and ideas. For one particular session the group split into three sections to discuss medical public relations problems in small, medium, and large communities. Reports were given on projects of medical societies which have proved effective in actual tests. The result was an increased knowledge of successful county public relations solutions and assurance of sincere cooperation among physicians and operating personnel.

TEXAS FIGHTS POLIOMYELITIS

The 1950 record of approximately 2,800 cases of poliomyelitis in Texas represented almost one-tenth of the total of new cases in the United States. Physicians are well aware of the importance of fighting the disease. They know that contributions to the March of Dimes campaign, which will last from January 15 to 31, are essential, but beyond that they realize that medical research into the cause and cure of poliomyelitis is vital.

In the last three years Texas received more than $350,000 outside aid as provided by the National Foundation for Infantile Paralysis. Furthermore, Texas was second only to Michigan among states whose March of Dimes funds were depleted by the 1950 epidemic, receiving more than half a million dollars. Of the national goal of $50,000,000 for 1951, Texas must raise at least $2,500,000.

During the first ten months of 1950 Texas received the following out-of-state aid: 12 resident physicians, 235 nurses, 11 nursing advisors, 22 physical therapists, 87 respirators, 61 hot pack machines, and more than 100 beds and cribs. Teams of experts from Pennsylvania and Colorado set up treatment facilities in Houston, San Antonio and Gonzales. This effective massing of forces offers striking proof that pooling available resources on a national level is necessary to guard any community where poliomyelitis may strike in epidemic proportion.

If an incidence rate of 70 times the normal epidemic load which occurred in a small Virginia town were to prevail in Texas, Houston would have 11,400 cases, Dallas 8,430, Fort Worth 4,940, and Austin 1,855. Considering that the 2,800 cases for 1950 strained the facilities of the state, Texas physicians can be grateful that such is not true.

An important first step in the development of a state plan to insure good and complete care for children and adults with poliomyelitis is the recently formed Texas State Polio Planning Committee. It consists of representatives from ten health and welfare organizations on the state and national level. The Committee has considered problems which include (1) a program to maintain contact between patient and family, (2) increased bed capacity for acute, convalescent, and long-term cases, (3) adequate equipment for hospitals, (4) additional professional personnel for hospitals, (5) adequate housing and maintenance for recruited personnel, (6) competent supervision and in-service training for emergency personnel recruited for

hospitals, and (7) continuity of total care from the acute period through convalescence and after care.

Texas physicians, like other Texas citizens, must be willing to assume the burden of caring for the state's own poliomyelitis cases and to provide added funds for research. They may find that an insurance policy covering poliomyelitis may be the answer for their own families and for some of their patients and friends. Such policies have relatively low premium rates and insure either individuals or families up to $5,000 for each person. Reliable insurance agents can give details about policies with various companies who offer poliomyelitis coverage.

The success of the 1951 March of Dimes campaign is important, but the campaign is only one aspect in the fight against poliomyelitis in Texas.

ARMY PRAISES STATE MEDICAL ASSOCIATION

A letter of commendation from Major General R. W. Bliss, Surgeon General of the United States Army, has been received by Dr. William M. Gambrell, president of the State Medical Association.

General Bliss recently toured the Korean battle fronts. His own observations that the sick and wounded servicemen are receiving the highest quality medical care coincided with the opinions of General Douglas MacArthur and the Secretary of the Army, Honorable Frank Pace, Jr. The Surgeon General asserts that the Army's good showing cannot be explained on the basis of its entering the period of increased military activity with medical service at full strength and highly trained in field medicine but that rather the reverse was true. Not an insignificant part of the successful medical care in Korea, he believes, can be attributed to the excellence of the Army's professional residency training program instituted with the assistance of leaders in civil medicine at the close of World War II.

More important, he maintains, was the Army's ability to furnish at least one regular Army combat medical officer with proved ability to all combat units of battalion strength or larger. According to General Bliss, this action was possible through the activities of such societies as the State Medical Association which made continuously available the civil medical assistance necessary to support the nation's military needs.

In the letter General Bliss commented as follows:

"Colonel Liston, Surgeon of our Fourth Army Area, has informed me in glowing terms of the magnificent job done in Texas in regard to manning of Recruiting and Induction Main Stations, procurement and assignment, and the making of physicians available for the accomplishment of the thousands upon thousands of physical examinations of inductees rapidly recalled to the military service. He claims that he made his problems known to you and to your executive committee and, from that point on, the doctors of Texas, under your guidance, shouldered the job. Needless to say, it was a most creditable performance.

"I want you and the members of the Medical Society to understand that, without your help, the medical service of the U. S. Army could never have hoped to meet its mission," he continued. "I shall be deeply pleased if you will convey my gratitude and the gratitude of the Medical Service of the Army to all those doctors who contributed to the success of this most demanding medical undertaking."

CURRENT EDITORIAL COMMENT

THE CHALLENGE OF CARDIOVASCULAR DISEASE

More than 637,000 deaths annually in the United States from cardiovascular diseases account for about 44 per cent of all deaths. Approximately 9,000,000 Americans have heart disease; of these 500,000 are elementary and high school children. An estimated 152,100,000 workdays are lost each year because of

This department of the JOURNAL *presents editorial comments on current items pertaining to the science, art, and practice of medicine, contributed by members of the State Medical Association and scientists closely associated with the medical profession of Texas. Invitation is hereby extended to any member of the State Medical Association of Texas to submit such discussions for this department. The discussions should not be more than 500 words in length.*

diseases of the heart and blood vessels. This is the challenge of cardiovascular disease.

Equally as challenging is the individual patient. When a physician makes a diagnosis of organic heart disease, he realizes that in the care of the patient he has begun a losing fight. In the earliest stages he offers general advice: "avoid strenuous activities; live sensibly; watch your weight; don't worry, the heart is a wonderful organ." Before too long symptoms develop and the doctor braces the patient with digitalis or other drugs, restriction of usual activities, more rest, and more encouragement. Again, before long more urgent symptoms force a retreat. Bedrest, low sodium intake, diuretics, and other well known measures are brought to the front and the line is stabilized. But not for long. All too soon increasing pressure bends the line and retreat begins again. Now, there are left no more reserves—no more in the heart and no more in the hands of the one trying to help the heart. Then only surrender remains. Not infrequently the enemy strikes suddenly with overwhelming power, and surrender occurs before the doctor can mobilize his forces.

This is not to minimize our present efforts. Our forces are better trained and more efficient than they ever have been, and we are able to hold the line longer than ever before. But present day efforts are not enough. Much more education and research will be needed before the course of cardiovascular disease can be reversed or its development prevented. The control of heart disease is a great challenge to every physician and layman.

GEORGE W. PARSON, M. D., President,
Texas Heart Association,
Texarkana, Texas.

401 East Fifth Street.

ORIGINAL ARTICLES

ANATOMY OF THE HEART AND GREAT VESSELS

Angiocardiographic Study

J. E. MILLER, M.D., Dallas, Texas

THE recognition and understanding of pathologic changes in any structure depend upon a knowledge of the anatomy and physiology of this structure. It is immediately apparent from a review of the various articles on anatomy of the heart and great vessels, however, that the anatomy appears different, depending on the author's interests.

The anatomist studies the heart and great vessels of a person who long since has been static. The structures are fixed and entirely different from the heart and great vessels in which the clinician or radiologist is interested. The pathologist also sees a static cardiovascular system through which blood is no longer coursing. His opening into the chest of necessity disturbs cardiac anatomy. Study of the removed heart does not reveal the relationship of the living parts. The surgeon, on the other hand, studies a dynamic cardiovascular system. Nevertheless, thoracotomy to some extent disturbs the anatomic relationships of these viscera. The clinician must study the living, dynamic person who is intact.

On many occasions I have seen a pathologist assign a lesion to a location several centimeters away from its position as demonstrated radiologically. This can be interpreted only to mean that the pathologist places the lesion as he sees it following the change that has occurred in this organ after living relationships have been disturbed. On one occasion when a surgeon indicated that I was mistaken in placing the aorta to the right of the esophagus, a perusal of the roentgenograms indicated that there could be no mistake and that the aorta was to the right of the esophagus.

Read before the Section on Radiology, State Medical Association of Texas, Annual Session, Fort Worth, May 2, 1950.

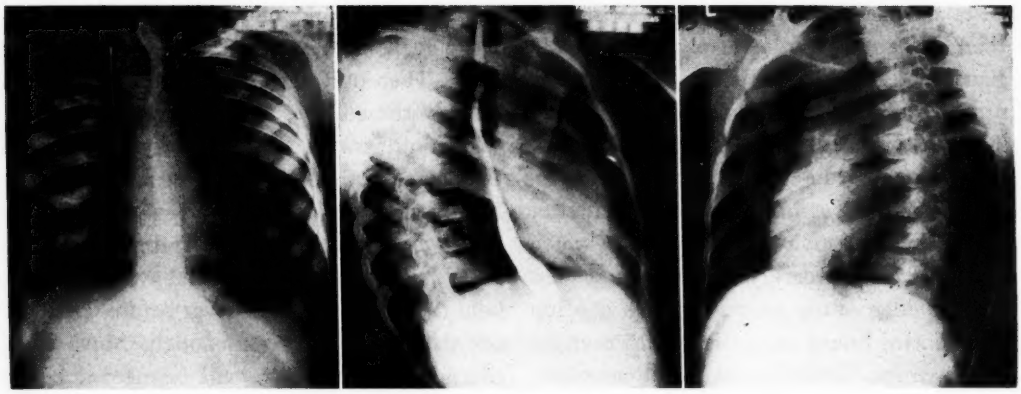

FIG. 1. Postero-anterior and right and left oblique roentgenograms of chest with barium in esophagus represent conventional examination.

When apprised of the fact that there could be no mistake, the surgeon dissected further and found that the aorta actually was to the right of the esophagus. With a collapsed lung and through a thoracotomy there must be of necessity some change in relationship of the intrathoracic structures. It is my opinion that the roentgen method where feasible is the best way to study anatomy.

Percussion gives an approximately correct idea of the position, size, and shape of the heart, but this information may be obtained more precisely by roentgenologic examination. Another attempt to substantiate and enlarge the knowledge obtained by the roentgen method was made by Dressler,[1] who developed an ingenious technique of transfixing the heart by means of passing needles through the anterior chest wall soon after death. The anterior thoracic layers were then removed one by one. This technique, however, proved subject to the same discrepancies as other methods of studying cardiac topography. Writers using such techniques have been careful to point out that the correlation offered by such studies is approximate at best.

The advent of a practical method of visualization of the heart and great vessels with development of angiocardiography by Robb and Steinberg[3] in 1938 elucidated many enigmas concerning the heart and great vessels. Angiocardiography, although not entirely physiologic, appears to offer the best method of studying anatomy of the heart and great vessels, despite the fact that if one accepts the recognized radiographic picture of the cardiac silhouette obtained by other methods, several points of variation can be found. Since it is my opinion that angiocardiography is the most accurate method of studying cardiovascular anatomy, the following review is based on this method.

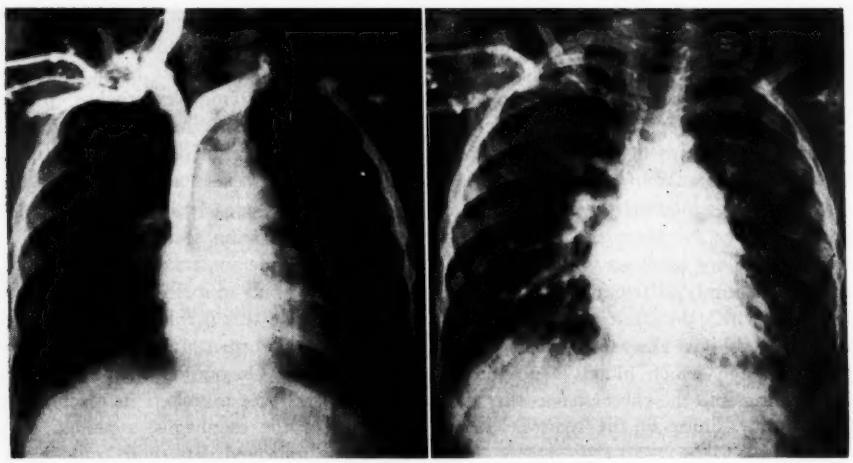

FIG. 2. Angiocardiograms showing *(left)* the right atrium and ventricle and *(right)* the position of the left atrium to the right.

ANGIOCARDIOGRAPHIC REVIEW

Angiocardiography affords a means of determining the size and shape of the heart. It is possible to differentiate between cardiac hypertrophy and chamber dilatation. Angiocardiograms, unlike conventional radiography, demonstrate clearly and unequivocally which chamber is larger in most cases of ventricular enlargement. Valves can be located. The position of the chambers as well as the great vessels can be ascertained. Most important, the border forming structures of the cardiac silhouette can be clearly demonstrated.

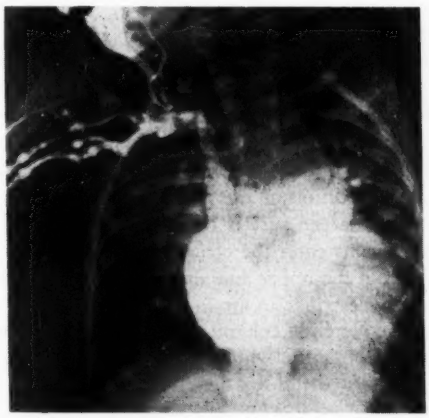

FIG. 3. The pulmonary conus well visualized far inside the left cardiac border.

Frontal Projection.—In the postero-anterior projection the superior vena cava occupies the upper right portion of the cardiac silhouette. The right mediastinal border above the aortic arch is formed by the right innominate vein. The right atrium forms the lower portion of the right border of the heart extending down to the dome of the diaphragm. The right atrium and ventricle occupy about one-half of the frontal cardiac area. This is at variance with many treatises on cardiac anatomy as studied by other means.[2] The inferior vena cava at times may form the lowermost portion of the right cardiac border. The ascending aorta normally does not form a part of the right cardiac border. With elongation and tortuosity of the aorta as seen in older persons it may extend lateral to the superior vena cava.

The right ventricle being situated deep within the cardiac silhouette is far removed from the left cardiac border. There are many references in the angiocardiographic literature to the effect that the pulmonary conus does not form a part of the left cardiac border.[5] It is a centrally located structure and in my experience has never formed a portion of the heart border on the left. In a postero-anterior film of the chest the bulge along the left cardiac border which is frequently referred to as the "pulmonary conus" is formed on occasion by the main pulmonary artery. It is more frequently formed, however, by the left pulmonary artery.

The left atrium is a circular structure which in reality is situated more to the right than to the left. It does not form a part of either cardiac border. The left ventricular shadow forms the left half of the heart. Many times the constriction between the left atrium and the left ventricle can be fairly well visualized, and it marks the location of the mitral annulus.

The left cardiac border is formed by the aorta above, the pulmonary artery segment, and then the left ventricle. Above the cardiac apex on the left border is a point on either side of which the pulsations are opposite. This so-called point of opposing pulsation does not represent any constant or definite landmark of cardiac chambers. A great deal of significance

FIG. 4. The right atrium and ventricle occupy only half of the cardiac silhouette in this frontal projection. The prominence on the left border is the pulmonary artery and its left branch.

which it does not deserve is given to this point throughout the literature. Measurements made from this point do not have any anatomic significance. The descending aorta sometimes forms the border of the composite silhouette in the middle cardiac segment.

Right Anterior Oblique Position.—In the right anterior oblique position the superior vena cava can be traced into the right atrium. This chamber rests upon the diaphragm and does not encroach upon the retrocardiac space. The outflow tract of the right ventricle representing the pulmonary conus is better visualized in this view. It is noted that the prominence

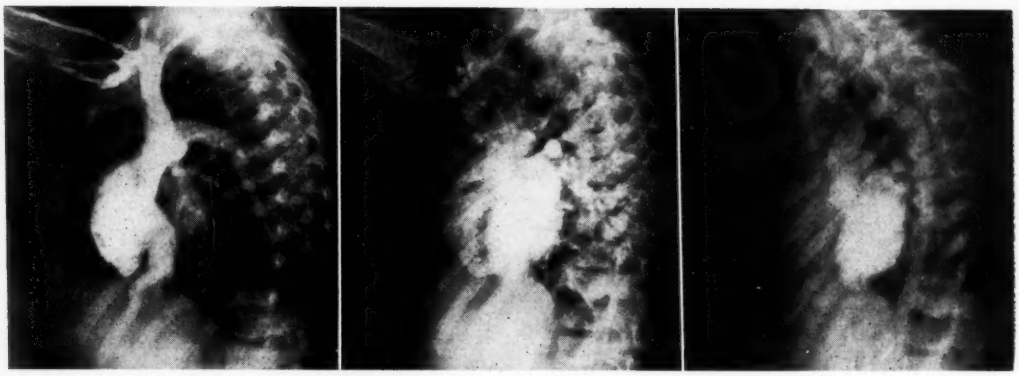

FIG. 5. The heart chamber and vessel anatomy as shown in the left anterior oblique position.

above the anterior border of the cardiac silhouette represents the main pulmonary artery and again is not the pulmonary conus. The patient must be viewed in a true lateral projection before the right ventricle actually forms a border of the cardiac silhouette. Occasionally cusps of the pulmonary valves as well as other valves can be recognized. In this position a rather large portion of the heart anteriorly does not opacify, indicating that it represents the left ventricle. The left atrium is situated posteriorly and occupies a slightly higher plane than the left ventricle. The left atrium forms the upper portion of the posterior border of the cardiac silhouette in this projection. It is in this area that the radiologist looks first to find slight enlargement of the left atrium, most commonly seen in mitral stenosis and patent ductus arteriosus.

Left Anterior Oblique Position.—In the left anterior oblique position it is difficult to recognize the right atrium and right ventricle as separate chambers. The interventricular septum or posterior boundary of the right ventricle is convex anteriorly. The superior vena cava enters somewhat posteriorly. The right pulmonary artery is foreshortened and the left visualized in its full length. The left atrium is well visualized in the upper portion of the heart posteriorly and forms the upper portion of the posterior heart border. The lower and larger part is the left ventricle. The pulmonary veins enter the atrium posteriorly. The aorta arises from the upper and anterior portion of the left ventricle, describes an arc in the thorax, and descends along the vertebral column. The left pulmonary artery produces the aortic windows by passing between the two limbs of the aortic arch.

SUMMARY

The most accurate method of studying cardiac anatomy in the living person is angiocardiography. Discrepancies in cardiac anatomy as found in textbooks and many articles are pointed out. The pulmonary conus is situated deep in the cardiac silhouette

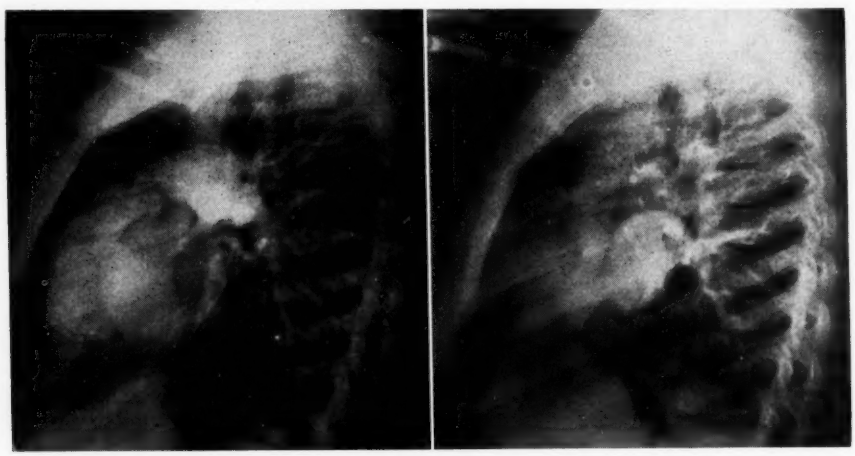

FIG. 6. The heart chamber and vessel anatomy as shown in the left lateral position.

and never forms a portion of the left border of the heart in the postero-anterior projection. This prominence, "the pulmonary artery segment," is formed frequently by the left pulmonary artery and on occasion by the main pulmonary artery. In the postero-anterior projection, the right side of the heart does not occupy the major portion of the cardiac silhouette. In the right anterior oblique projection, the left ventricle occupies a considerable portion of the cardiac silhouette anteriorly.

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DIAGNOSIS OF HEART DISEASE

Conventional Roentgen Examination

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THE examination of the heart and great vessels has always been of interest to the roentgenologist, but it has become of much greater importance in the recent developments of cardiovascular surgery. Accuracy in diagnosis of cardiovascular diseases is in direct ratio to the index of suspicion. Most cardiac lesions can be suspected if not accurately diagnosed in the conventional roentgen examination, but the roentgen observations must be correlated with clinical findings and a relatively small number of patients will require the expensive, time-consuming specialized procedures for an accurate diagnosis.

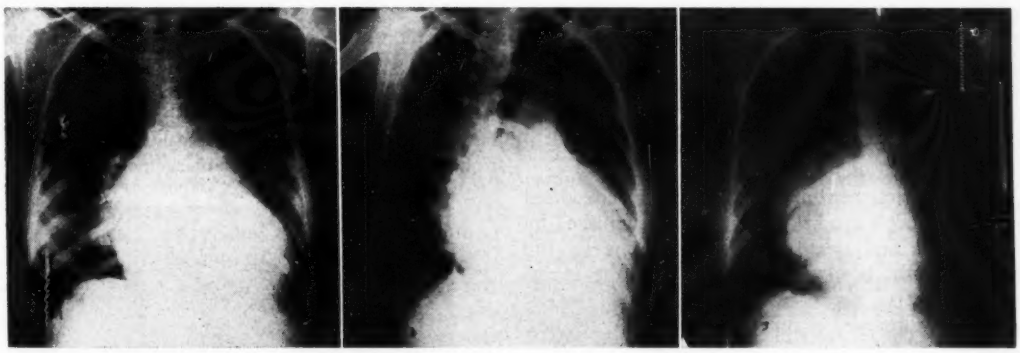

FIG. 1. A woman, aged 28. Clinical diagnosis: rheumatic heart disease with mitral stenosis and insufficiency; aortic stenosis with insufficiency. The roentgenograms show marked enlargement of all heart chambers.

CHEST DEFORMITIES

In general, the chest wall determines to a large degree the configuration of its contents. Variations in build and stature cause significant changes in the heart and great vessels. These features have been discussed by Roesler,[4] Sussman,[7] and others, but it should be mentioned that respiration causes changes in cardiac size and the relative position of the diaphragm influences the cardiac configuration. In old persons generalized emphysema results in an apparently small heart. Great caution must be exercised in comparing films made in the vertical and horizontal positions. Elevation of the diaphragm is probably the greatest factor in the horizontal position but also the heart appears enlarged.

Cardiac displacement is a factor to be observed with caution in deformities of the spine. In kyphoscoliosis, which occurs most frequently with the deviation to the right, the heart is displaced to the left side of the chest cavity. There is associated rotation of the heart which makes an examination at different angles necessary in order to secure a true perspective of the heart. Severe dorsal kyphosis causes a shortening of

Read before the Section on Radiology, State Medical Association of Texas, Annual Session, Fort Worth, May 2, 1950.
An abstract of the discussion of this paper follows the next article.

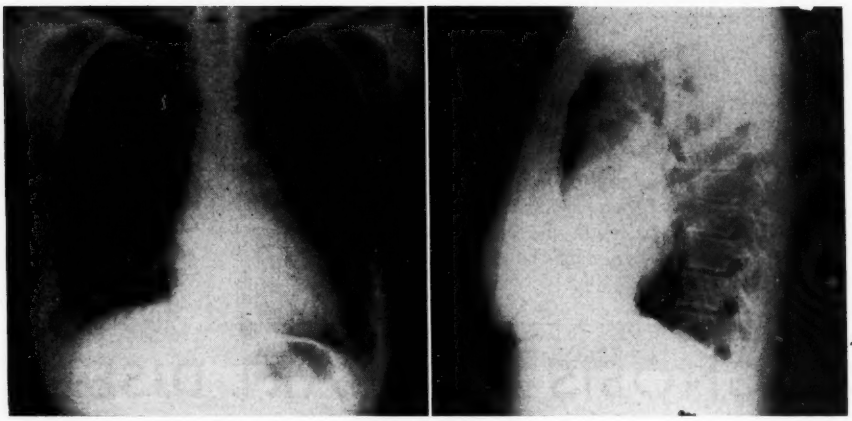

FIG. 2. A woman, aged 46. Clinical diagnosis: rheumatic heart disease with mitral stenosis. Generalized cardiac enlargement is evident in the roentgenograms. There is relative prominence of the pulmonary segment and left auricular enlargement.

the heart shadow and forces it to a transverse position. In these conditions the esophagus frequently appears abnormally placed in relation to the heart. The heart may be depressed in the deformity of the sternum and the left border frequently shows apparent enlargement.

DISEASES

The heart is often displaced with pleuropulmonary disease. Since the heart and great vessels are suspended in the mediastinum, contraction of one lung by fibrosis causes displacement. In pneumothorax shift as well as rotation of the heart can be observed. Obstructive emphysema usually causes a change of the position of the heart, and this position may vary in different phases of respiration.

Cardiac enlargement may be caused by a generalized loss of tone. This condition is not well established by the clinician and is difficult to determine by the roentgenologist; however, fluoroscopically the examiner may observe variations in the pulsations. The enlargement is observed in chronic degenerative diseases, metabolic diseases, and in severe anemia. In these conditions the enlargement may be due to hypertrophy as well as dilatation of one or several chambers. There is also a physiologic enlargement as greater demands are made on the circulation.

Enlargement of a single heart chamber is uncommon, but from the roentgen point of view certain basic changes are rather characteristic for the various chambers and a composite of these findings is usually necessary in the average case of cardiac enlargement. Left ventricular enlargement appears as an elongated left border with rounded caudad displacement of the apex. There is also posterior enlargement in the lateral projection. In extreme cases, the heart is enlarged to the right and then differentiation from right ventricular enlargement is difficult. Right ventricular enlargement is characterized by anterior bulging and obliteration of the retrosternal space on the lateral projection. In contradistinction to left ventricular enlargement, the apex is frequently displaced upward. There is usually associated widening of the heart to the left and there is elevation of the pulmonary segment on the left border of the heart in the posteroanterior projection. Enlargement of the left atrium is best observed in the right oblique position, where it encroaches on the retrocardiac space, frequently with compression of the esophagus in a limited area. This is the only chamber which, because of the increased density, can be recognized within the heart shadow. As the enlargement increases it appears on the right border of the heart and frequently forms a second right upper cardiac curve. Enlargement to the left

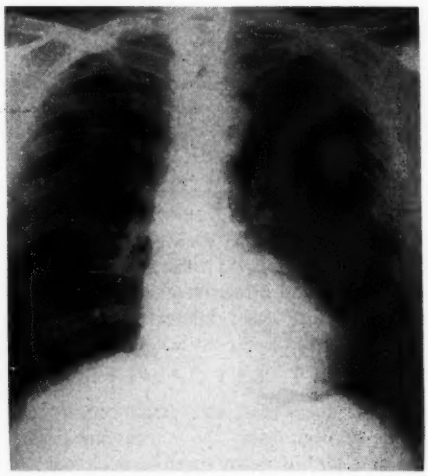

FIG. 3. A white man, aged 56. The roentgenogram of the chest shows mild ventricular enlargement. The patient had syncopal attacks with chest pains for six months and died with acute pulmonary edema. Postmortem examination revealed calcified aortic stenosis.

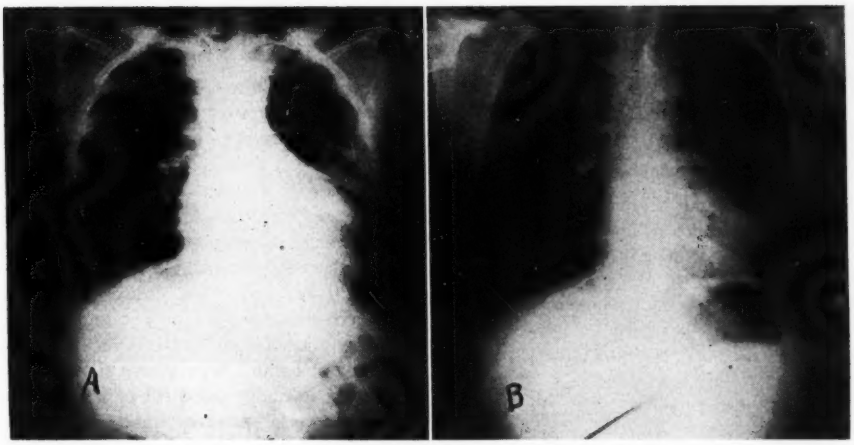

FIG. 4. A boy, aged 18. The patient had had cyanosis since birth and had been operated on for tetralogy of Fallot. Repeated pulmonary hemorrhages usually occurred with heavy exertion. The roentgenogram on the left shows marked cardiac enlargement with pulmonary congestion. The roentgenogram on the right shows some reduction in heart size.

occurs in the area of the left auricular appendage. These combined factors cause rotation of the heart to a relatively right oblique position with widening of the angle at the tracheal bifurcation and elevation of the left main bronchus. The right atrium enlarges to the right and a differential point is that it does not encroach on the esophagus when visualized in the right oblique position.

The changes in the left side of the heart almost always are due to rheumatic fever, syphilis, and hypertensive arteriosclerotic heart disease. The changes are dependent upon the site of greater involvement. In case of mitral disease, it is usually rheumatic fever. Aortic valvular disease, except the stenotic type, which is nearly always from rheumatic fever, is most frequently syphilitic or arteriosclerotic. In "pure" mitral stenosis the left ventricle is not enlarged but the left auricle is extremely large. If mitral insufficiency predominates, the left ventricle is also large. Aortic valvular disease is manifested by an enlarged ascending aorta and left ventricular enlargement. The changes demonstrable are not always consistent with the valvular changes while the heart is compensating. The roentgen examination may show calcifications of heart valves, and these are more frequently the stenotic type. Tricuspid involvement rarely is demonstrated as an independent condition but usually is associated with other valvular disease.

Coronary thrombosis, as such, usually causes no change in the contour of the heart. Changes depend upon antecedent disease and the degree of heart failure. There may be a change due to significant pericardial effusion. Infarctions may cause aneurysms or may become calcified.

The exact amount of fluid required in the pericardium is not known, but it is estimated that 300 cc. may be roentgenologically significant.[4] The time consumed for the development of the effusion is important because a chronic process allows the pericardium to conform to the fluid with changes in the silhouette. General changes to be observed are obliteration of the posterior inferior recess on the lateral projection; obliteration of the superior recess, especially in the recumbent position, with foreshortening

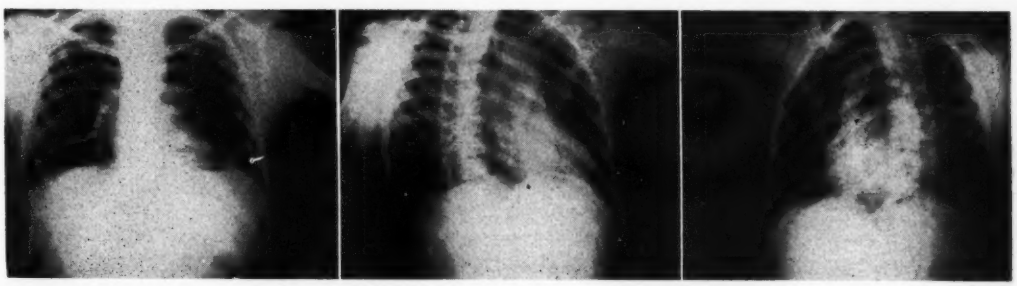

FIG. 5. A girl, aged 9. Clinical diagnosis: coarctation of the aorta. The blood pressure was 160/80. The roentgenograms reveal a suggestion of scalloping of the right seventh rib, but it is not diagnostic. The aortic knob is small. Clinical diagnosis was confirmed by surgery. Blood pressure after surgery was 110/70.

of the vascular trunk; and a loss of the normal area of the heart with rounded enlargement of the heart shadow. There may be a variation or loss of the pulsations by fluoroscopy.

CONGENITAL ANOMALIES

A number of congenital heart diseases have become amenable to the more recent developments of surgery. It is estimated that 80 per cent of these cases

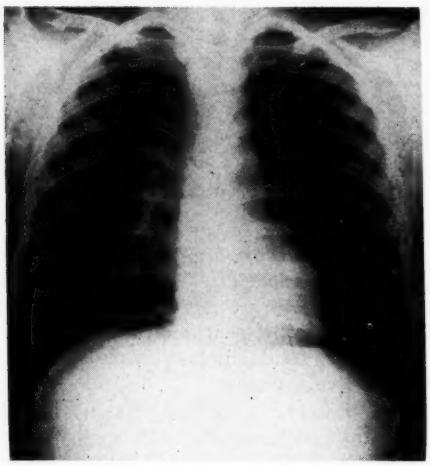

FIG. 6. A boy, aged 12. The patient was referred because of heart murmur. Clinical diagnosis: coarctation of the aorta. The blood pressure was 176/110. The roentgenogram shows a small aortic knob, and there is a small scallop on the inferior border of the right seventh rib and an indication of one on the left. Diagnosis was confirmed by surgery, and the postoperative blood pressure was 124/70.

can be accurately diagnosed by an astute clinician with the assistance of the conventional roentgen-ray examination.[5] Angiocardiography and heart catheterization have contributed much in the study of cardiac physiology; consequently these procedures are now required only in selected cases.

In infancy the heart may show a globular enlargement. This is of little value for a specific diagnosis. It is also noteworthy that major anomalies may be present without any significant roentgen change. From a practical viewpoint about 70 per cent of cyanotic infants who survive beyond 2 years have tetralogy of Fallot.[1] In this condition the hilar markings are thin, the pulmonary segment on the left border of the heart is depressed, and the lower left pole of the heart is prominent in the frontal projection as a result of right ventricular enlargement. It has been demonstrated that about 20 per cent of a group of cases showed no roentgen abnormalities except right ventricular enlargement in oblique projections in the presence of tetralogy of Fallot.[6] A persistent right-sided aortic arch is present in about one-fifth of the cases of this disease.[2] Since the primary indication for the Blalock anastomosis is the presence of a tetralogy of Fallot, any gross variations in the roentgen picture such as marked cardiac enlargement or large pulmonary vessels are indications for the more specialized diagnostic procedures.

Eisenmenger's complex consists of the same basic defects as tetralogy of Fallot but there is no pulmonary stenosis. This changes the roentgen picture so that the cardiac enlargement is more marked, the pulmonary artery is prominent, and the lung fields show a normal or prominent vascular structure. It is important to differentiate this condition since it is not amenable to surgery.

Patent ductus arteriosus is not diagnosed by the roentgen findings, but this examination can be suggestive or confirmatory. There is usually moderate enlargement of the pulmonary artery. Fluoroscopically, the heart, aorta, and pulmonary artery appear hyperactive. The heart usually shows moderate to marked enlargement. The cardiac enlargement is most prominent in the left ventricle. Stauffer[6] reported an enlarged left auricle in about one-fourth of the cases, a ratio which makes it frequent enough to be significant in the diagnosis.

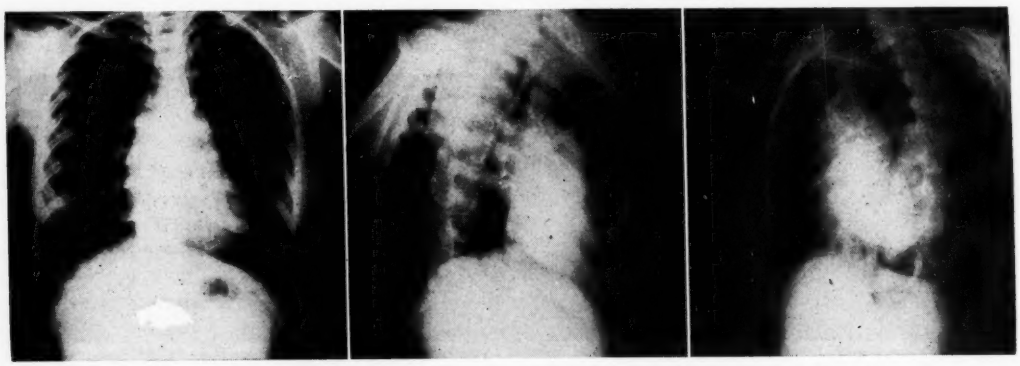

FIG. 7. A woman, aged 21. Clinical diagnosis: intra-auricular septal defect. The roentgenograms show primarily an enlargement of the left auricle and a mild enlargement of the left ventricle.

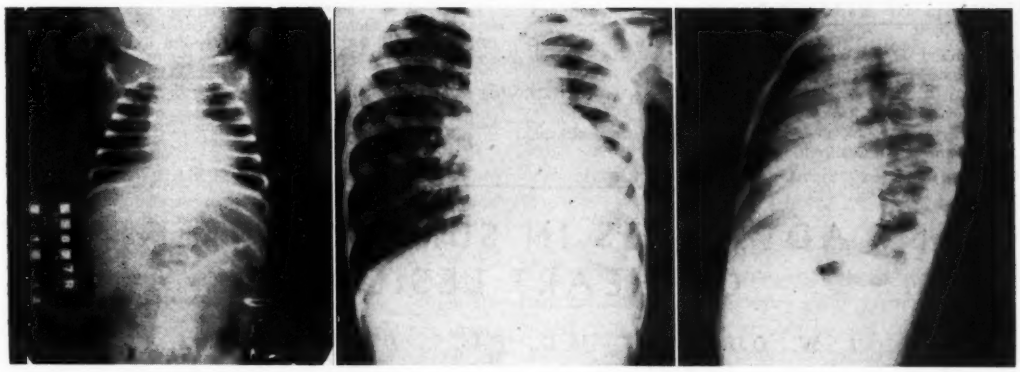

FIG. 8. The left roentgenogram is of a 3 weeks old girl who was having attacks of cyanosis. The heart is in relatively transverse position. There appears to be mild thymic hypertrophy. The other two films were made when the patient reached the age of 6 years. They show marked ventricular enlargement. Clinical diagnosis: intraventricular septal defect.

Coarctation of the aorta can be identified roentgenologically by the small aortic arch, dilated ascending aorta, prominent subclavian artery, and irregular notching of the inferior borders of the ribs.[3] The heart may be normal in size, but it frequently shows mild enlargement of the left ventricle. Notching of ribs is not always sufficient to be significant, especially in children, but the combined roentgen signs are usually adequate to support the clinical diagnosis.

Septal defects must be considered because there are some which have become amenable to surgery. If the interauricular defect is small, there may be no significant changes. If the defect is large, the heart is tremendously enlarged and globular in shape. The pulmonary artery is prominent and the aortic arch is frequently not visualized. With an interventricular defect, generally the heart is not as markedly enlarged even with a large defect. In cases of a small defect the heart contour may be normal. The aortic arch is prominent and the prominent pulmonary artery is not unlike that in case of interauricular septal defect.

SUMMARY

The conventional roentgen examination is an important adjunct in the diagnosis of heart disease.

Variations in the bony thorax and the pleuropulmonary structures are significant in the differential diagnosis.

Cardiac enlargement may be generalized or limited to individual heart chambers.

The discussion of congenital heart disease is limited primarily to lesions which may be amenable to surgery. An accurate diagnosis can be made in the great majority of cases by a thorough clinical examination and the conventional roentgen examination.

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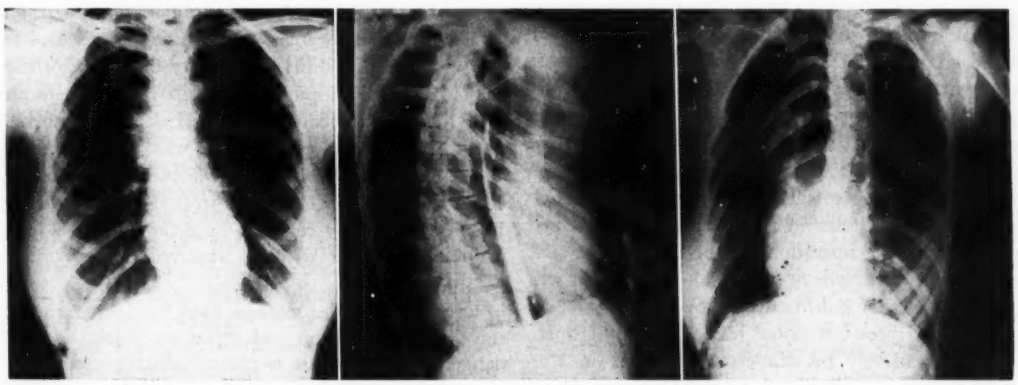

FIG. 9. A woman, aged 28. Routine chest roentgenograms show marked enlargement in the area of the ascending aorta and to the right. Barium in the esophagus shows no deviation. The roentgen impression was fusiform enlargement of the aorta. The patient had no symptoms relative to the chest. Surgical exploration revealed a cirsoid aneurysm of the superior vena cava. The azygous vein was also involved in this aneurysmal dilatation. The enlargement extended from approximately the midportion of the superior vena cava down to the atria. There was no growth present.

DIAGNOSIS OF HEART DISEASE—Barsh—*continued*

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2010 Broadway.

RECENT ADVANCES IN SURGICAL TREATMENT OF HEART LESIONS

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THE first attempt at surgical treatment of heart disease was made by Doyen in 1913. This was an unsuccessful operation in a case of congenital pulmonic stenosis. During the next fifteen years, 11 operations were reported by other authors, 7 being done by Cutler and Beck. Nine of the 12 patients died at operation or shortly after. Of the 3 survivors, all were reported improved; 2 had only digital dilatation of the stenosed valve. With such discouraging initial efforts, it is little wonder that interest in cardiac surgery lagged for the ensuing decade. Then in 1938, Gross of Boston revived this interest by the successful ligation of a patent ductus arteriosus, stimulating the development of a new and brilliant field of surgery which has resulted in the successful treatment of thousands of patients with congenital malformations of the heart and in the promise of the early conquest of acquired lesions of the heart.

SURGERY ON GREAT VESSELS

Two congenital lesions, patent ductus arteriosus and coarctation of the aorta, may be completely corrected; a third group, characterized by cyanosis, prominent in which is the tetralogy of Fallot, now may be symptomatically relieved by operative means. Gross deserves credit not only for first successfully closing a patent ductus arteriosus but also for developing an operation for correction of coarctation of the aorta, although Crafoord of Stockholm in October, 1944, performed the first successful operation for this lesion. The first operation for treatment of the tetralogy of Fallot was done by Blalock of Baltimore in November, 1944. At present, these three congenital lesions are being operated on extensively by numerous surgeons in this country and abroad and several modifications of the original operations have been developed.

Read before the Section on Radiology, State Medical Association of Texas, Annual Session, Fort Worth, May 2, 1950.

Patent Ductus Arteriosus

Simple ligation of the patent ductus arteriosus was soon abandoned by Gross because of 10 per cent recurrence in the early cases so treated. For a time, Gross tried various methods, such as wrapping with cellophane, to stimulate fibroplasia about the ligated ductus. This was soon given up for division and suture of the divided ends of the ductus, which is the method Gross has now used in more than 500 cases with an over-all mortality of less than 2 per cent. To replace the four clamp method of Gross, Potts has devised a pair of clamps with rows of tiny teeth which prevent slipping by penetrating the adventitia. A courageous technique has been carried out by Crafoord when the ductus is short and wide. He completely occludes the aorta with clamps proximal and distal to the ductus, and with a curved clamp impinging on the wall of the pulmonary artery, he divides the ductus and closes the defects in the walls of the major vessels by suture. He has kept the aorta occluded for as long as twenty-five minutes without ill effects.

Although division of the ductus is desirable as a means of complete and final elimination of the shunt, it is considered by many surgeons too hazardous because of the possibility of uncontrollable hemorrhage should there be slipping of a clamp. Blalock maintains that ligation is adequate provided that multiple ligatures are used and placed in a way that completely obliterates the lumen of the ductus throughout its length. A total of 161 cases treated by this method have been recently reported by Scott from the Johns Hopkins Hospital with no recurrences and only 2 deaths.

Regardless of the method used for obliteration of the shunt by division and suture or by ligation, the danger of hemorrhage is greatest during the preliminary dissection and isolation of the ductus. Especially on its posterior aspect where the enveloping fibrous tissue is dense and exposure difficult, a thin walled ductus may be easily torn and serious hemorrhage

result. To avoid a possibly fatal outcome from such an accident, it is important, as emphasized by Jones, first to free the aorta proximal and distal to the ductus so that hemorrhage may be controlled while the ductus is rapidly isolated, divided, and sutured.

Rather than to adopt a routine procedure for all cases, it seems more logical to employ the method suited to the requirements of each case. When the ductus is narrow and has sufficient length for accommodating three or more suture-ligatures as used by Blalock, this method is safe and effective. When the ductus is wide, even multiple ligation does not insure complete obliteration of its lumen throughout its length, and division and suture should be done, using any of the clamp methods described by Gross, Potts, and others. When the ductus is exceptionally short, or when there is only a fistulous opening between aorta and pulmonary artery, Crafoord's plan of clamping the aorta may be used or the Potts aortic clamp may be applied. I have found that the pulmonary end of the short ductus or fistula may be easily handled by application of the screw clamp with curved blades developed by Blalock for isolating a portion of the auricle in the transposition operation.

Hundreds of operations for patent ductus arteriosus have now been done in various medical centers over this country and abroad. Mortality statistics have varied considerably as surgeons become more experienced. A 2 to 5 per cent mortality should now be expected.

Coarctation of Aorta

Coarctation of the aorta is the second congenital cardiovascular malformation for which corrective operation is possible. Fortunately, in most cases only a short segment of the aorta is stenosed at or near the level of the ligamentum arteriosum and distal to the left subclavian artery. This situation is favorable for resection of the stenotic area and end-to-end anastomosis as originally described by Crafoord and by Gross in 1944. The success of this operation depends on the presence of adequate collateral circulation carrying blood to the trunk and lower extremities, making possible the complete occlusion of the aorta while the resection and anastomosis are carried out. Between 5 and 15 years is the optimum age for performing this operation, and in this age group mortality should be no more than from 5 to 10 per cent. Many operations are now being done on older patients but with a higher mortality rate because of increased sclerosis and friability of the vessel walls and because of changes in heart and brain attendant upon the long standing hypertension.

When the coarctation involves a segment too long to allow resection and end-to-end anastomosis, it may be possible to utilize the greatly enlarged left subclavian artery to bridge the defect, as suggested by Blalock but first performed by Clagett. Another method for bridging the long gap has recently been developed and used with success by Gross. The stenosed segment is resected and replaced by a homograft of aorta taken from a cadaver and kept at low temperature in a suitable culture medium until ready for use.

Lesions Causing Cyanosis

The third group of congenital heart malformations treated successfully by surgery is characterized clinically by cyanosis of greater or less degree—the so-called "blue babies." Most frequently found in this group is the tetralogy of Fallot, in which there is always an association of four lesions: (1) stenosis or atresia of the pulmonary artery, (2) interventricular septal defect, (3) displacement of the aorta to a position overriding the septal defect, and (4) hypertrophy of the right ventricle. Other less frequent lesions causing cyanosis are tricuspid atresia, single ventricle, and truncus arteriosus with bronchial arteries.

For the entire group of cyanotic lesions, the operation developed by Taussig and Blalock affords relief of the symptoms produced by lowered arterial oxygen saturation. This condition is the result of (1) diminished blood flow to the lungs and (2) mixing of venous and arterial blood entering the aorta from the heart. The operation does not attempt to alter any of the malformations of the heart and great vessels but simply produces an artificial patent ductus arteriosus or aortic-pulmonary shunt which has the result of increasing blood flow to the lungs. By this means the level of arterial oxygen saturation is raised appreciably, often approaching normal amounts, so that there is marked improvement in symptoms such as dyspnea, low exercise tolerance, dizziness, fainting, and convulsions. Cyanosis is partially or completely relieved.

The essential feature of the Blalock operation is an anastomosis between a systemic vessel, usually one of the subclavian arteries, and the pulmonary artery on the same side. An end-to-side anastomosis is preferred so that blood from the aorta may enter both lungs, but numerous variations of the operation are possible, depending on size and anatomic relationships of the vessels. The operation is usually done on the right side of the chest to take advantage of the favorable angle made by the subclavian branch of the innominate artery.

Another operation for the same purpose was devised by Potts, Smith, and Gibson in 1945. This is the direct side-to-side anastomosis of the aorta and the left pulmonary artery, made possible by an ingenious clamp which isolates a lateral segment of the aorta while blood continues to flow past this segment into

the descending aorta. In about 20 per cent of patients with the tetralogy of Fallot, the aorta turns to the right and passes downward on the right of the spine. In this group, either the Blalock or the Potts operation is technically difficult if attempted on the right side, and the operation is best done on the left side by the Blalock method. When the aorta descends on the left, the Potts operation is preferable, especially in small children and infants because of the small size of the subclavian artery. By this choice of operations, it is always possible to enter the left side of the chest and avoid the necessity for accurate diagnosis of the location of the aorta before operation.

Since 1944, more than 950 operations of the cyanotic group of heart lesions have been done at the Johns Hopkins Hospital by Blalock and his associates with an over-all mortality of about 17 per cent. Since these patients in general are much poorer operative risks than those with patent ductus arteriosus or coarctation of the aorta, the higher mortality is to be expected. In the most favorable age group, 3 to 10 years, mortality is only 7 per cent. In numerous smaller series now being reported by other surgeons in this country and abroad, mortality rates closely approximate the Johns Hopkins figure. Although the risk of surgery is great, it seems entirely justified by the excellent results obtained by the survivors and consideration of the undeniably poor prognosis without operation. Without surgery these children seldom live past the age of 10 or 12 years and many die in infancy. As noted, the best age for operation is probably from 3 to 10 years, but surgery should be done earlier if symptoms are especially severe, since death may occur at any time. I have operated successfully on an infant 6 weeks of age. Of 78 cyanotic patients on whom I have operated, 12 died, resulting in a mortality rate similar to the Johns Hopkins figure.

Just what the future holds for these patients after operation is uncertain. So far, only a few have developed complications such as cardiac failure or subacute bacterial endarteritis. The majority are enjoying fairly normal physical activity and development, some for as long as five and one-half years. It is to be hoped that the surgically produced lesion and the congenital malformations will balance one another and allow the heart to function without significant strain for a long life expectancy.

INTRACARDIAC SURGERY

The operations being done daily in ever increasing numbers on patent ductus arteriosus, coarctation of the aorta, and cyanotic congenital heart lesions are actually directed at the great vessels. Meanwhile, a few surgeons are beginning again to experiment with intracardiac operations for treatment of both congenital and acquired lesions.

Stenotic Heart Valves

Since 1945, Smithy, Harken, Bailey, and Blalock in this country, Murray in Canada, and Brock in England have reported small series of 10 to 20 patients operated on for stenotic heart valves. Most of these have been cases of mitral stenosis complicating rheumatic endocarditis, but several patients have had congenital pulmonic stenosis. Pure pulmonic stenosis occurring without the septal defect and overriding aorta present in the tetralogy of Fallot is not helped by producing a shunt. If the stenosis is in the valves and not in the infundibular portion of the artery, the pulmonic channel can be opened by valvulotomy. This has been done in 20 cases by Blalock using Brock's operation. Brock also has attempted to treat the tetralogy of Fallot with pulmonary valvulotomy instead of producing an extracardiac shunt. In the 10 cases he reported, death resulted in 5. These operations have been done by introducing through the wall of the right ventricle a cutting instrument called a valvulotome. The stenotic valve is cut open by the valvulotome in an entirely blind procedure. Blalock has recently operated on several cases of tetralogy of Fallot by this method, but believes the shunt operation is a better procedure.

Transposed Aorta and Pulmonary Artery

Another congenital malformation, transposition of aorta and pulmonary artery, has been treated surgically with some success by Blalock. Since there is complete transposition of the great arteries without transposition of the great veins, the systemic and pulmonary circulations are actually separate, a condition incompatible with life unless some type of shunt is also present. In patients who survive, there is always found a shunt such as patent ductus arteriosus or interauricular or interventricular septal defect. Even with a shunt, life expectancy is short, averaging 19 months. There is extreme cyanosis, dyspnea, and physical disability.

For treatment of this lesion, Blalock at first attempted various arterial and venous shunts with no success. He then developed a technique for producing an interauricular septal defect and used it in 12 cases with 9 deaths. The 3 survivors were only slightly benefited. In the next 16 cases, the interauricular septal defect was made in combination with an anastomosis between the subclavian artery and pulmonary artery. Of these patients 4 died, and all of the 12 survivors were improved, though not to the degree that is obtained by the operation for tetralogy of Fallot.

Septal Defects

Little has been accomplished in treating pure congenital septal defects, a rather common lesion. If the defect is small, it may not produce significant symptoms, but if large, there is considerable disability and short life expectancy. Murray in Toronto has suc-

ceeded in reducing the size of the large septal defects by passing wide mattress sutures of fascia through and through the heart in the axis of the septum and tying them with sufficient tension to bring the margins of the defects close together. By decreasing the amount of shunted blood, symptoms were improved. One of the 4 patients operated on died.

Mitral Stenosis

Although by far the most striking results have been obtained in surgical treatment of congenital malformations of the heart, an increasing number of successful operations are now being done for acquired heart lesions, especially mitral stenosis. In 1945 Harken in Boston and Smithy in Charleston revived the earlier efforts at treating mitral stenosis by removing a segment of the stenosed orifice with the valvulotome. In Harken's first report, 2 operations had been done with 1 death. In 3 subsequent cases, there were 2 deaths. Both survivors were benefited. Smithy, himself a victim of rheumatic valvular heart disease, died after having performed 8 operations for mitral stenosis with 2 deaths. Bailey in Philadelphia has developed a technique seeking to avoid mitral regurgitation which results from valvulotomy. In this operation, the valve is palpated by a finger introduced through an incision in the auricular appendage, and a back-cutting knife is passed through a metal guide on the finger to incise both commissures. The valve cusps are said to open and close with a considerable degree of efficiency.

MECHANICAL HEARTS

Thus far, all intracardiac operations have been done without benefit of vision. Cardioscopes of limited use in inspecting the condition of diseased valves have been experimented with by Graham, Murray, and others, but none has proved of value in performance of the operative procedure. In order that circulation to vital organs, especially the brain, be maintained through the course of an operation, it has been necessary for the heart to function as a pump and for the chambers to be filled with blood at all times. This requirement has blocked the development of accurate repair of abnormal or damaged valves or closure of septal defects, but at the same time has stimulated experiments with mechanical heart-lung systems capable of maintaining circulation and oxygenation of the blood while heart chambers are empty. Such systems have been devised and used successfully with experimental animals.

Dennis in Minneapolis, Gibbons in Philadelphia, and Crafoord in Stockholm use symptoms which perfuse only the brain with artificially oxygenated blood for operations on the empty heart up to thirty minutes in duration. They find that no significant alterations occur in other organs deprived of blood during that period of time. Jongbloed in Utrecht, Holland, has constructed a more efficient but highly complicated apparatus which maintains circulation of artificially oxygenated blood to almost the entire body including brain, spine, heart muscle, liver, kidneys, and endocrine organs, and bronchial circulation to the lungs. Only venous blood does not enter the heart and the pulmonary circulation. With this apparatus the heart chambers in dogs have been kept empty of blood, or nearly so, for as long as two hours with full recovery afterward. So far, there is no report of such an apparatus having been used in man. It seems highly probable that when technical difficulties are removed, this approach to the problem of intracardiac surgery will offer opportunities for startling new operations, and relief for untold thousands of sufferers from heart disease.

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ABSTRACT OF DISCUSSION

DR. GLADYS J. FASHENA, Dallas: These presentations highlight a subject whch is assuming increasing importance, not only because of the increasing morbidity due to heart disease, but also because advances in surgery offer promise of dramatic benefit for conditions which have hitherto been considered hopeless.

In the proper evaluation of cardiac disease determination of the physiologic abnormalities which are present and determination of the functional capacity of the diseased heart are of prime importance in many instances in making the decision for or against operative intervention. For example, determination of the exact anatomic lesion present in a cyanotic child, although obviously important, may be difficult or even impossible with the tests now at our disposal. However, if the physician can determine to his satisfaction that the pulmonary blood flow is decreased and that the pressure relationships are such that a shunting type of operation like the Blalock or Potts procedures will enable mixed venous and arterial systemic blood to reenter the pulmonary circulation, the chances are overwhelming that the patient will be improved by surgery regardless of the exact anatomic defect. On the other hand, if such a patient exhibits marked cardiac enlargement, the physician must weigh the probability of improved oxygenation obtainable by surgery against the danger of cardiac failure which may develop as the result of the increased load upon the heart incurred by the shunt. My limited experience with congenital heart lesions has convinced me of the necessity of thinking not only in terms of the anatomic defect but even more importantly in terms of the pathologic physiology involved.

In passing, I must comment upon the low regard in which Dr. Paul White apparently held the technique of roentgen examination of the cardiovascular system. I believe he stated that roentgen examination contributes about 5 per cent to a complete cardiovascular study. Certainly in the field of congenital heart disease this percentage is a gross underestimation. My belief is that history and careful roentgen and fluoroscopic study contribute more than 75 per cent to the diagnosis of congenital cardiac lesions. Excepting patent ductus arteriosus, auscultation of the heart in patients having congenital defects contributes little to the diagnosis and prognosis, and I consider careful roentgen and fluoroscopic examination an important part of the clinical study necessary accurately to appraise the patient's condition.

Dr. Barsh has succinctly outlined interpretation of the changes in cardiac contour which occur in disease. The only thing which I would like to add in this conneetion is that conventional interpretations are not always accurate. Angiocardiography has taught us many exceptions to the rule. For example, although outward and downward displacement of the left cardiac border usually signifies left ventricular enlargement, it has been my experience that such a contour is occasionally seen with predominant right ventricular enlargement. Similarly, in the left anterior oblique view, although physicians ordinarily interpret forward displacement of the anterior border as signifying right ventricular enlargement and backward displacement of the posterior border as signifying left ventricular enlargement, I have found occasional patients in which the reverse was true. The moral is (1) that the physician must interpret all findings in the light of the total body of knowledge which he can amass concerning the patient and (2) that there must be the closest kind of cooperation and discussion between the clinician and the roentgenologist.

There is little I can add to Dr. Duckett's comprehensive discussion. The results of the surgical approach to cardiac disease eloquently speak for themselves. There is no more heartwarming sight than the transformation of a woebegone, physically incapacitated invalid into a happy, healthy, normal-looking child. What the future may hold for amelioration of heart diseases of all types can only be glimpsed at present, but the expectation of greater things to come should be a challenge to every physician to attempt more complete and accurate cardiac diagnoses.

Heart Patients and Jobs

A critical need for an expanded rehabilitation program to return the heart patient to physical, mental, and economic usefulness, especially in the light of current demands for manpower mobilization, has been emphasized by two New York physicians. Writing in the December issue of *Modern Concepts of Cardiovascular Disease,* Drs. Joseph G. Benton and Howard A. Rusk of the Department of Physical Medicine and Rehabilitation of the New York University Bellevue Medical Center call for an active and vigorous rehabilitation program.

"The available effective manpower reservoir is at present 2½ million compared to 8 million in 1950," the doctors write. "Should the national mobilization proceed at an increasing tempo, it is anticipated that industry and the armed forces in a mobilization program will rapidly absorb this group. Further mobilization will, of necessity, have to draw on the large number of disabled and the older age groups for its labor pool."

Employers must be educated to accept cardiac patients who have been rehabilitated and who are capable of holding suitable jobs, the physicians assert. Housewives, "the largest segment of the working cardiac population," should not be overlooked in the rehabilitation program, they add.

Medical Fellowships Offered

Ten fellowships in the medical and allied sciences fields supported by the Graduate College of the University of Illinois to be awarded to qualified scholars will be announced April 1, 1951. Applications must be received by February 15, 1951, and forms may be obtained from the Associate Dean of the Graduate College, University of Illinois, 808 S. Wood St., Chicago 12.

Applicants need not have completed clinical internships. Minimum requirements are: bachelor's and medical degrees; bachelor's and dental degrees; bachelor's degree in pharmacy and master of science degree. Stipends range from $1,200 to $3,000 per calendar year; appointees are exempt from tuition fees.

Nothing is more completely proved than the fact that approximately one-half of all cases of significant tuberculosis have no symptoms, or symptoms so slight as to escape notice. —A. C. Christie, M. D., Pub. Health Rep., June 2, 1950.

MECHANISM AND MANAGEMENT OF MYOCARDIAL INSUFFICIENCY

Treatment of Cardiac Edema

GEORGE R. HERRMANN, M.D.; HUGH P. REVELEY, M.D.*; MILTON R. HEJTMANCIK, M.D.; and GEORGE IV H. C. HERRMANN, M.D., Galveston, Texas

THE hope of the good physician is to forestall and postpone the development of symptoms and signs of myocardial insufficiency. The clinical evidences of potential or latent asymptomatic heart disease are recognized and the diagnosis should be made long before heart failure develops. A clear understanding and prompt interpretation are desirable in any derangement of function which may insidiously develop in patients who have old healed heart lesions. In recent years great masses of new data have accumulated. A knowledge of the critical considerations is not only desirable but necessary for the scientific handling of patients.

The appearance of any of the cardinal symptoms, shortness of breath, cough, extreme weakness, pain in the chest or under the right costal margin, and swelling about the ankles, suggests that failure of the heart and circulation have developed. In a patient with the signs of structural damage in the heart, physicians are ever alert for any of the cardinal symptoms. They usually look for some confirmatory signs such as dullness and rales at the lung bases, an accentuation of the pulmonic second sound, engorgement of the neck veins, enlargement of the liver, and pitting of the dependent parts. We usually measure the venous pressure in the anticubital vein and find it increased while the circulation time is usually slowed.

Edema of the lungs, of the liver, and especially of the subcutaneous and interstitial tissue is the most disturbing complication, and its removal is the most pressing problem in the management of patients with heart disease and myocardial failure. Edema signifies excessive accumulation of extracellular fluid without definite bounding membranes to the point that there occurs swelling and pitting on pressure of the skin. The distribution need not be uniform as there may be collections of fluid due to local disturbances of the causative factors.

The principal components of edema fluid are sodium chloride and water, the content of protein depending on the conditions of formation. Regardless of type, the mechanism of edema has been attributed to disturbances in the normal relation of the hydrostatic and colloid osmotic pressure of the capillaries and tissue spaces. Increased vessel permeability rarely may be a contributing factor. These concepts of pressure gradients in the capillaries were brought forth by Starling[17] more than forty years ago and were recently reaffirmed by Peters.[10] However, there has come to be recognized an additional endocrine renal factor, which in some cases seems to be the most significant from the quantitative standpoint. Congestive heart failure represents, in its most usual form, edema of the type most amenable to treatment, and it is here that the new ideas have been most fruitful.

BACKWARD FAILURE VS. RENAL DYSFUNCTION

Within the past decade the traditional concepts of "backward congestive" and "forward circulatory" failure have been questioned by many clinical investigators and totally abandoned by some. Critical scrutiny has been directed toward the old view of increased pressure in the venous reservoirs draining toward the failing cardiac chambers as the fundamental mechan-

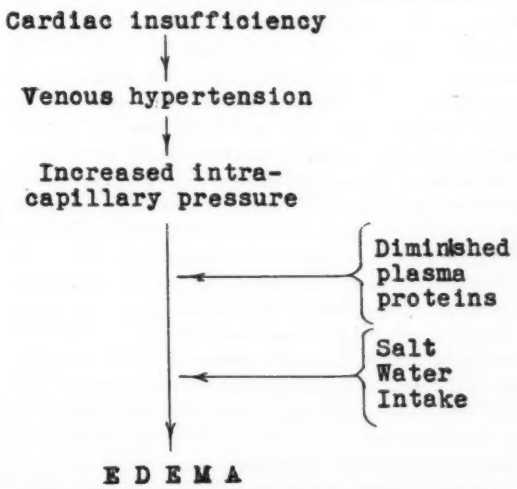

FIG. 1. Diagram showing the sequence of events in the classical backward failure theory of congestive failure.

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Supported by the H. H. Weinert Fund for Cardiovascular Research and a grant-in-aid from Campbell Products, Inc., New York, which supplied Thiomerin for this study.

**National Heart Institute Trainee, 1950-1951.*

Material presented before the Section on Medicine, State Medical Association of Texas, Annual Session, San Antonio, May 3, 1949, and before the First International Cardiological Congress, Paris, France, September 7, 1950.

ism underlying congestive failure. A sequence of events (fig. 1) based on simple mechanical disturbances in the central pump and great vessels has been shown inadequate to explain the complete picture.

Studies of the cardiac output in various types and stages of heart disease, by application of the direct Fick principle made possible by catheterization of the right side of the heart, have demonstrated that under certain circumstances congestive failure occurs with a cardiac output increased above normal. In general, the low or high ventricular minute volume output depends upon the type of cardiac involvement (table 1). The type of heart disease with an absolutely decreased cardiac output usually leads to relatively early decompensation and in general responds promptly to sup-

TABLE 1.—*Maximum Cardiac Output Response in Diseased Hearts.*

Decreased	Increased
Acute myocardial infarction	Aortic regurgitation
Coronary atherosclerosis	High pulse pressure aortitis
Acute rheumatic myocarditis	Systolic hypertension, diastolic hypotension
Chronic rheumatic mitral stenosis	Hyperthyroidism
Low pulse pressure	Arteriovenous shunt
Hypotension	Congenital lesions
Hypothyroidism	Anemia
Heart tamponade	Beriberi
Pericardial constriction	
Shock, hemorrhage	

portive treatment, whereas the "high output" failure occurs late and is refractory to therapy. Starling's "Law of the Heart" is still applicable as the failing heart can increase its output by dilating under the increased strain. However, even with adequate augmentation of oxygen supply there is a critical length of the myocardial fiber beyond which an increase leads to actual fall in the cardiac output.

A number of observations have shown that the back pressure caused by weakness of the central pump cannot explain all of the associated phenomena. In studies of cardiac tamponade by us following those of Cohnheim,[1] the venous pressure could be made to rise, but there was also a fall in arterial pressure. There thus was evident a fundamental difference from congestive heart failure, in which arterial pressure is constant or increased. Constructed mechanical models[18] can be made to show a rise of venous pressure under a maintained arterial pressure only if the total volume of fluid in the system is increased. Starr[18] furthermore was unable to reproduce right ventricular failure in animals by destroying the right ventricular wall with cautery. His faith in the traditional concepts was shattered, particularly when he contemplated the fact that mercurial diuretics and salt deprivation restored circulatory equilibrium. The factors of rest and load relief were not considered.

In the patients of Warren and Stead,[19] the feeding of salt and the withholding of mercurial diuretics, after their vigorous use had produced a "dry weight," resulted first in an increase in weight and then in a rise in venous pressure. These observers concluded that salt and water retention is the most important primary factor in the development of congestive heart failure. Reichsman and Grant[13] reemphasized the primary importance of venous pressure rises. They showed in patients with atrial fibrillation that after withholding digitalis, venous pressure rise preceded any gain in weight or edema. The English observers would attribute this venous pressure rise to constriction of the veins.

Merrill[8] has emphasized the importance of sodium and water retention in the mechanism of high output failure, in which the minute volume is considered reduced relatively, with blood supply to the kidney disproportionately low as the result of demands by other tissues. That the kidney of congestive heart failure is unable to excrete sodium normally has been demonstrated by Futcher and Schroeder[2] and by Reaser and Burch.[12] In addition to decreased glomerular filtration, sodium retention is also due to increased reabsorption of sodium in the renal tubules. There may occur increases in the antidiuretic hormone and adrenal cortical hormone as a result of inadequate circulation to the pituitary or adrenal gland.

There has thus been suggested and derived a new concept of congestive failure which emphasizes extra-

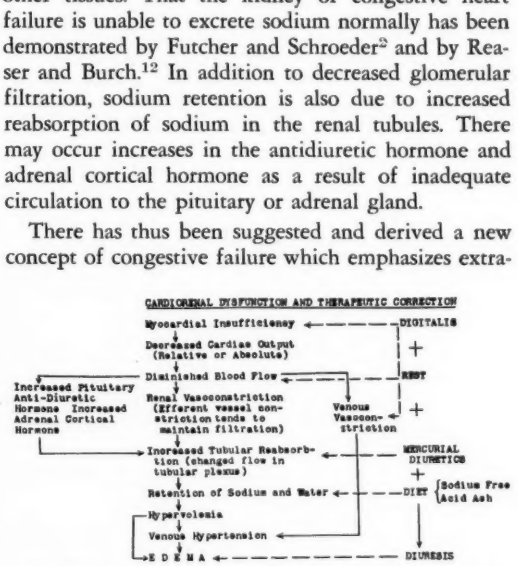

FIG. 2. Diagram illustrating the sequence in a new concept of congestive failure which emphasizes extracardiac factors and showing points of effectiveness of therapeutic measures.

cardiac factors in a sequence of events as diagrammed in figure 2. Points of effectiveness of the therapeutic measures are also shown in this diagram.

It is obvious that the primary source of difficulty is the insufficiency of the myocardium itself. Compensatory increases in the oxygen consumption and in the venous pressure develop to help maintain circulatory equilibrium. Myocardial weakness may be defined as the state in which the normal mechanical work of the heart is not maintained. Cardiac output determinations up to now have been accurately done only by a few expert clinical physiologists, and for the most part at rest. Cardiac catheterization probably will never become a routine procedure which

the general internist will use with his patients. A recent study[20] has suggested the changes of body electrical capacity as a means to record cardiac volume. However, the cardiac systolic and diastolic volumes in the kymoradiogram may be subtracted for a practical estimate of the cardiac output. The diastolic volume as shown radiologically in the frontal plane may be considered as a rough index of heart muscle fiber length, which may be correlated with mechanical work.

DIAGNOSIS

For the diagnosis of congestive heart failure, physicians must still depend on an assembling of clinical data such as engorgement of veins, increased venous pressure, cardiac enlargement, engorgement of the liver, dependent edema, and oliguria. Some one of the reliable criteria of heart disease is usually demonstrable. There may be evidence of cardiac enlargement or ausculatory signs such as presystolic gallop rhythm or murmurs indicative of organic valvular lesions. Myocardial damage may be revealed by the electrocardiogram by QRS and axis changes, ST segment shifts, conduction defects, or serious mechanism disorders. Diagnoses of aortic atheromatosis, coronary insufficiency, chronic nephritis, diabetes, myxedema, and exophthalmic goiter are frequently associated with and result in myocardial damage when not controlled.

Although the role of the heart may have been overly minimized in recent discussions, the reevaluation of the importance of the extracardiac factors of congestive failure has placed therapy on a sounder footing and made it more effective. Even Harrison[5] in reaffirming the backward failure theory admits that the renal factor may be regarded as quantitatively the most important in cardiac edema. Accordingly, we have come more and more to emphasize therapy directed at dissipation of edema by the kidneys.

TREATMENT

To restore compensation in the failing circulation, therapeutic measures should first be directed against the underlying cause. It is thus desirable to increase cardiac output and to decrease tissue needs in order that there may be a balance between the two. This can be accomplished by (1) the decrease of the demands on the cardiovascular system by lessening activity and by providing adequate sedation; (2) the use of digitalis to increase the efficiency of the myocardium; (3) the administration of coronary vasodilators to bolster myocardial oxygenation and nutrition; and (4) the correction of abnormal states of metabolism or structural abnormalities which make the output inadequate.

At times the circulatory situation may assume the serious form of an emergency, with acute left ventricular failure and pulmonary edema. Under such circumstances, positive pressure oxygen, morphine sulfate, phlebotomy, and intravenous digitalization may be necessary. Mechanical drainage of embarrassing accumulations of edema fluid may be required. Cardiac arrhythmias, which reduce myocardial efficiency, should be abolished, and this accomplishment alone may improve the circulation enough to reestablish equilibrium.

Rest.—Some of the strain upon the circulation is relieved by rest, but it should be accomplished in a position most comfortable to the patient, usually the sitting position, in order to avoid an upward shift of fluid to waterlog the lungs. Morphine relaxes spasm, relieves exhausting effects of dyspnea, and allays anxiety. Demerol better relaxes the spasm of cardiac asthma and does not tend to produce nausea or vomiting; however, it has the greater danger of addiction.

Prolonged strict confinement to bed is to be avoided, and the hazards of excessive stay in bed have been recently reemphasized. Passive exercises should be administered to all bed-ridden cardiac patients. Anticoagulant therapy is definitely indicated if thromboembolic phenomena develop and even has been suggested as a prophylactic routinely in congestive failure.[21] However, if accurate prothrombin control is not available, Dicumarol is dangerous.

Diet.—A condition of relative acidosis of the body is desirable, since acid-yielding foods and salts call forth the sodium base from the tissues and thus aid in its excretion. We have found that some cardiac patients have an absolute as well as a relative oligoalbuminemia, and such patients with cardiac edema need a generous protein intake. Schemm[14] has restricted the sodium intake in the diet to 800 mg. or less daily. Although he has advocated forcing of fluid intake to 6 or 8 liters a day, such high levels have been found by others[9] to be of questionable value. It would appear that a low sodium diet providing 200 mg. of sodium daily with sodium-free water as desired would be a rational method of therapy. By using Lonolac milk in the Karell diet sodium intake may be reduced to 50 mg. a day. In some areas the sodium content of the city water may be unduly high, such as 34 mg. per 100 cc. in Galveston, and in such cases distilled water should be used. Schemm also recommended intravenous injection of from 2 to 4 liters of 5 per cent glucose in patients with heart failure, but circulatory overload and pulmonary edema has been noted after such therapy. Supplemental vitamins are essential as in any patient who is on a restricted diet.

Digitalis.—Although digitalis is classically the sovereign drug for the treatment of congestive heart failure, some have concluded that it is unnecessary since

edema can be eliminated with diuretics and adherence to a low sodium diet. Digitalis increases the efficiency of the cardiac muscle. It slows the ventricular rate in atrial fibrillation and should always be used in atrial fibrillation with failure. It seems logically indicated in all cases of congestive failure unless there is some complicating factor, such as paroxysmal ventricular tachycardia, contraindicating it. The choice between the oral and parenteral preparations depends upon the urgency with which the effect is desired. Purified glycosides now in many places have largely supplanted the powdered leaf. However, the single-dose oral method of digitalization using Digitoxin, 1.2 mg. has not been adequate in many of our cases. After this dose we usually complete and maintain digitalization by the use of a good standard 0.1 Gm. (1.5 grain) tablet of powdered digitalis leaf daily.

Diuretics have been employed in the treatment of congestive failure for several decades. In the early 'twenties the mercurial diuretics displaced the xanthines because they were more effective. Mercurials have come to be used more and more. They act by decreasing reabsorption of sodium and water in the tubules, while the xanthines increase glomerular filtration and may alter tubular reabsorption. It was found that when the mercurial was given with the xanthines, there was augmented diuresis and less tendency to irritate the kidney. Some serious and a few fatal reactions following intravenous injections have been reported in recent years, and increased myocardial irritability and acute cardiac depression have been observed.

The older mercurial diuretics were primarily for intravenous use, but Mercuhydrin was developed as a well-tolerated drug that could be given intramuscularly. Still less toxic is Thiomerin, which can be given subcutaneously. Premedication with ammonium chloride or other acid salts enhances the action of the new mercurials but usually is not required. Too rapid and excessive diuresis by the use of mercurials may lead to the dangers of sodium depletion, particularly when the sodium intake is being rigidly curtailed.

Symptoms such as suddenly increasing dyspnea, weakness, muscle cramps, nausea, restlessness, apathy, shock, and coma may be caused by excessive sodium depletion.[16] The blood nitrogen rises, and the presence of the condition may be confirmed by finding a low blood sodium or chloride level. The administration of double normal saline solution relieves the symptoms; sodium supplementation should be guided by blood electrolyte studies. We have observed only 2 patients who may fall into the category of suffering from sodium depletion. This is probably because of the difficulty in obtaining a low sodium intake in Galveston.

EXPERIMENTAL STUDIES

Recently we have followed the effects of parenteral mercurials, intravenous aminophylline, and intravenous digitalis on patients in congestive failure. Plasma and urine sodium and chloride levels, urine volumes, blood specific gravity and hematocrits, and plasma volume determinations with Evans blue dye were made. The venous pressure was determined repeatedly using a constant zero level.

CASE 1.—J. T. was in severe congestive failure with generalized anasarca due to hypertensive and arteriosclerotic heart disease. He had never been treated before. Three hours after the administration of 2 cc. of Thiomerin subcutaneously, diuresis ensued at the rate of 16 cc. of urine per minute; sodium excretion was 49 mg. per minute although the sodium concentration of the urine fell. As diuresis proceeded, the urinary sodium level paralleled the urine flow. Plasma sodium levels did not fall significantly during the early hours of diuresis; however, after cardiac compensation was restored, the plasma sodium level was 132.5 milliequivalents per liter in contrast to the admission level of 145 milliequivalents per liter.

Venous pressure fell slowly as mercurial diuresis proceeded. After a loss of 1,300 cc. of urine over a period of seven hours it fell to 24 cm.; when a total of 8,300 cc. of urine, containing 23.5 Gm. of sodium, had been excreted over eleven hours, the venous pressure had fallen to 15.4 cm.

Pugh and Wyndham,[11] employing catheterization of the right side of the heart, obtained similar results when they found that a pure mercurial diuretic had no effect on right atrial pressure until diuresis was well under way.

Following mercurial diuresis, which still maintained a urine flow three times the level prior to treatment, patient J. T. was given 1.6 mg. of Cedilanid intravenously. There was immediate conspicuous fall in venous pressure. Ten minutes after injection of a full digitalizing dose of Cedilanid the venous pressure fell from 15.4 cm. of saline solution to 12.5 cm.; at sixty minutes it was 11 cm.; three hours later it was 9 cm.

McMichael[7] and his group have demonstrated the dramatic lowering of right atrial pressure following administration of 1.5 mg. of Digoxin.

Maximal rate of diuresis following digitalization did not occur until three hours had elapsed. Sodium concentration of the urine fell initially as from dilution but after six hours the concentration was 138 milliequivalents per liter and the sodium excretion rate was 19.5 mg. per minute. Full digitalization appeared to produce a more massive and more prolonged diuresis than the mercurial, but the effect of Thiomerin was probably not dissipated when digitalis was injected. In the thirty hours after giving Thiomerin 8,300 cc. of urine containing 23.5 Gm. of sodium were excreted; in twenty-eight hours digitalis effected the excretion of 10,300 cc. of urine, which contained 25.7 Gm. of sodium.

In 2 other patients 0.8 mg. of Cedilanid administered intravenously did not lower the venous pressure within three hours and produced no significant diuresis.

CASE 2.—M. E. was in moderately severe congestive failure due to hypertensive and arteriosclerotic heart disease and was on a maintenance dose of digitalis. After administration of 500 mg. of aminophylline intravenously the venous pres-

sure promptly fell from 24 cm. to 13 cm. of saline solution. Within ten minutes diuresis started at the rate of 3.8 cc. per minute in contrast to the control rate of 144 cc. in ten hours. Sodium excretion rose from 0.8 mg. per minute to 10 mg. per minute and the urinary sodium concentration increased from 52 milliequivalents per liter to 120 milliequivalents per liter. Maximal diuresis of 6 cc. of urine per minute was attained one hour later and slowed after three hours to 2.3 cc. per minute. At three hours venous pressure had begun to rise, being 14 to 16 cm. of saline solution; twelve hours later it had risen to 18 to 20 cm. of saline solution. In twelve hours after aminophylline administration 1,200 cc. of urine which contained 3.6 Gm. of sodium were excreted. On the next day the patient was given a mercurial diuretic and in twenty-four hours excreted 4,000 cc. of urine, which contained 10 Gm. of sodium.

Howarth and others[6] have shown that aminophylline produces a sharp drop in right atrial pressure within five minutes; they noted an immediate rise in cardiac output which passed off within ten to fifteen minutes. Greene, Paul, and Feller[3] demonstrated the general circulatory effect of aminophylline when they noted a fall in venous pressure following intravenous injection of the drug. Sinclair-Smith and others[15] have expressed the belief that aminophylline has a twofold effect modified by the degree of cardiac decompensation: it directly affects the circulation in increasing cardiac output thus augmenting glomerular filtration rate and renal plasma flow, and it diminishes reabsorption of filtered sodium by the renal tubules.

We believe that changes in circulatory dynamics induced by aminophylline are reflected in the changes in plasma volume. In patient M. E. intravenous aminophylline immediately produced an apparent rise in plasma volume and the venous pressure fell. However, within twenty minutes the plasma volume fell below the expected level and continued to fall as diuresis proceeded. When Thiomerin was given to patient J. T., there was merely a gradual fall in plasma volume secondary to the loss of urine as diuresis continued. We were unable to detect a significant rise in plasma volume following a mercurial diuretic at any time in the patients observed.

Some observers have noted an initial hemodilution of the blood during mercurial diuresis, but most investigators found a hemoconcentration with mercury. Pugh and Wyndham[11] noted no change in cardiac output until it increased after diuresis was well under way following injection of a pure mercurial. The major action of mercury is limited to the kidneys; any effects on circulatory dynamics are secondary to the primary renal effect. Herrmann has maintained that prompt primary sodium filtration and consequent temporary immediate lowering of serum sodium is the trigger mechanism that calls forth sodium and water from the tissues after injection of a mercurial diuretic.

Sodium excretion is increased by all diuretics. Aminophylline and the mercurials effect an early rise in sodium excretion but mercurials produce a much more prolonged and pronounced effect. Digitalis produces a slower but persistent rise in urinary sodium concentration. Further studies may elucidate whether this augmented excretion of sodium is due to improved renal status or is primarily the result of altered circulatory dynamics as the failing heart promptly regains compensation.

ORDER OF THERAPY

In the management of congestive failure prompt determination of the venous pressure provides a firm base line for evaluation of treatment desired for most effective results. When there is marked elevation of venous pressure or a sudden rise in venous pressure as in acute left ventricular failure, slow intravenous administration of aminophylline is the emergency treatment of choice. It is in effect a bloodless phlebotomy. While the effect of aminophylline is being dissipated, intravenous digitalization should be carried out thus perpetuating and augmenting lowered venous pressure produced by aminophylline. Routine use of intravenous digitalis is not often desirable in the ordinary case of failure and should be avoided if the patient has had digitalis within three weeks; but it may be life-saving in acute pulmonary edema. If digitalis, preceded by aminophylline, does not appear adequately to mobilize the sodium and water, a mercurial diuretic such as Thiomerin should follow in the management of the edematous patient.

Such a program of therapy seems rational in the light of present knowledge. The conception of the reaction of the overloaded heart and its response to therapeutic procedures such as digitalization, aminophylline, and mercurial diuretics has been clarified, yet there are still many questions to be answered. Extensive and elaborate simultaneous studies of circulatory and renal dynamics are necessary to throw more light on the mechanisms involved and the establishment of the most rational, effective, and safe order of therapy.

SUMMARY

The mechanism of heart failure and edema is discussed, with particular reference to extracardiac, such as renal, factors.

Newer concepts of the routine supportive treatment are presented, step by step, and effectiveness of each is stressed.

Observations of the effects of aminophylline, digitalis, and mercurial diuretics on the venous pressure, plasma volume, and excretion of urine and sodium are described.

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EVALUATION OF MYOCARDIAL INFARCTION IN RAILWAY EMPLOYEES

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THIS study was undertaken to determine the ability to return to work of patients who have suffered myocardial infarction. We have been particularly concerned with safety to the patient and the public after resumption of employment and to this end have reviewed 205 consecutive cases at the Santa Fe Hospital in Temple, which receives only employees of the Santa Fe Railroad and various affiliated industries.

Of the 205 patients with myocardial infarction studied 34 were retired from active economic production at the time of infarction (fig. 1a). The 171 who were employed at the time of infarction and with whom we are primarily concerned represent various occupations of the railroad industry from major executives to laborers. Among the active employees were 158 white men, 3 white women, 4 Mexican men, and 6 Negro men. All of these patients have been accurately followed, most of them by periodic observation at the hospital (fig. 1b).

Of the 171 employees who by clinical and/or electrocardiographic means had the diagnosis myocardial infarction 4 per cent gave no history suggestive of myocardial infarction or an anginal equivalent. In only about one-half of the total group was there a prior history of angina. Various reports,[2, 11] as well as our own experience, indicate that the great majority of patients experience their attacks while at rest. These often are described as severe and rapid in onset. From our series 26 patients noted that attacks began while they were engaged in their occupations. Those having attacks during activity usually described them as becoming progressively intense over a period of several hours.

During the acute phase of the disease various observations are of prognostic value. The age of the patient seems to influence directly the mortality rate. Previous infarctions, cardiac enlargement, congestive failure, a persistent pulse rate of more than 120 or less than 60,[2, 5, 8, 10, 13, 15] prolonged shock, and embolic phenomena are associated with increased mortality. Temperature elevation, leukocytosis, and increased sedimentation rate are relative indicators of the severity of the attack.[5, 7, 8, 11, 13, 14, 15] Various changes of rhythm and conduction, such as auricular fibrillation, gallop rhythm, pulsus alternans, complete heart block, and multiple focal extrasystoles with the pulse rate more than 100, indicate a serious prognosis.[4, 5, 7, 8, 9, 13]

Twenty-two of the 171 patients died during the first month after the infarction, an immediate mor-

From the Department of Medicine of the Scott and White Clinic.
Read before the Texas Railway and Traumatic Surgical Association, May 1, 1950, Fort Worth, Texas.

tality rate of 12.8 per cent. Obviously, the discrepancies in the various reports in the literature concerning immediate mortality[1, 2, 11, 12, 16] result from many variable factors incidental to the groups selected.

At present 73 of these previously active patients have died. Of this group 30 per cent died during the first month, 45 per cent within one year, and 84 per cent within five years.

It is not our plan to attempt to outline the medical management of the patient during either the emergency period or early hospitalization. Late therapy, or treatment during the ambulatory phase of the disease, seems to be a compromise between overloading the damaged myocardium on the one hand and encouraging collateral circulation by increasing the local need for it on the other.[3] Ambulation is encouraged to avoid the muscle wasting, negative calcium and nitrogen balance, and prolonged weakness often associated with excessive periods of bed rest.[6] It has been our practice to allow these patients out of bed four weeks after the myocardial infarction if at that time there is no evidence of heart failure. The heart rate should be within normal limits, the electrocardiogram should show no evidence of the acute process, and the sedimentation rate should be at or near the normal level. Exercise is increased gradually but not to the point of exhaustion. We always strive to keep activity below the threshold that produces angina, dyspnea, or other cardial embarrassment.

After the patient's satisfactory convalescence, we recommend that he modify his physical activity in keeping with the above plan. A readjustment of his weight to normal levels, a program of general physical conservation, and in particular the avoidance of psychic trauma also are advised.

The prognosis of the patient who suffers the initial insult of myocardial infarction has undergone considerable change in the last few years. It has been shown that the postinfarction mortality rate for patients able to resume activity is higher than that of the corresponding average age and sex group. After two years, however, the mortality rate of the two groups shows about the same pattern.[2] Others[15] have reported that 25 per cent of persons who have had myocardial infarction have survived more than ten years.

One of the first patients in this series had an attack in 1932. The pain was severe and was associated with dyspnea and syncope. The patient soon returned to work and has continued working at his usual occupation. During this time he underwent a major surgical procedure without cardiac distress.

From our series approximately one-half of those who were able to return to duty did so within three months, and 4 out of 5 within six months. Generalized weakness, fatigue, psychoneurotic fixations, and causalgias of one or both upper extremities were among the most frequent complications delaying the patient's return to work later than four months.

Two-thirds, or 113, of those who were employed at the time of infarction subsequently returned to duty. Generally, it has been our practice to allow to return to work those persons who manifest no dyspnea or anginal pains in performing their usual duties and who otherwise show no evidence of uncompensated myocardial failure. If possible injury to the patient or to others may occur because of his sudden inability to perform his job, we advise that he be placed in such position only if someone can assume his duties immediately if necessary. Sudden or unexpected death usually is associated with coronary artery disease. It may be the first symptom of the disease and consequently is unpredictable. Our experience has shown sudden incapacity to occur infrequently in those who after suffering a myocardial infarction have a well

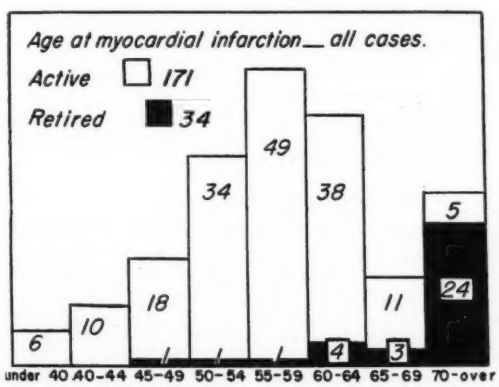
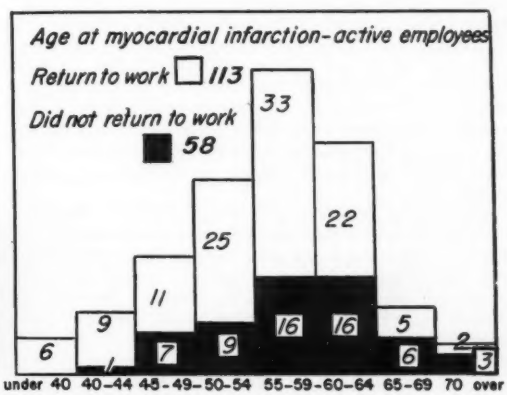


FIG. 1a. A graph demonstrating the distribution according to age of 205 consecutive patients with myocardial infarction.



b. A graph showing the distribution according to age of 171 of the 205 patients who were actively employed at the time of infarction.

compensated myocardium. Many patients undoubtedly would minimize or attempt to conceal their complaints if they thought discovery of such disease would result in dismissal from their jobs. It is our thought that medical counselors by helping to place these persons in desirable situations assure greater safety to all concerned. Of the total of 113 patients in our series who have been able to return to work, an incident in which personal or property damage was attributed to sudden incapacity was not noted. The patient, doctor, and employer by understanding each other's problems can effect safer conditions for more productive years.

SUMMARY AND CONCLUSIONS

The records of 205 patients with myocardial infarction are reviewed. Of these 171 were actively employed.

The mortality rate during the first thirty days after myocardial infarction in the active group was 12.8 per cent. Sixty-six per cent of these employees were able to return to work and, in most instances, to their former occupations. Fifty per cent of them returned to work within three months, and 80 per cent within six months after the infarction.

No instance of personal or property damage resulted from the sudden incapacity of a patient.

This study indicates that persons suffering from myocardial infarction have a better prognosis as to life and further employability than has been generally realized.

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Cortisone and ACTH in Arteriosclerosis

The possibility that arteriosclerosis will result when cortisone and ACTH are administered over a prolonged period has been demonstrated by three New York doctors.

"The factor of prolonged therapy assumes a particular importance because of the increased availability of these drugs," write Drs. David Adlersberg, Louis Schaefer, and Stanley R. Drachman in the November 11 issue of *The Journal of the American Medical Association.* They point out that it may be expected that many patients with chronic disease will now receive long courses of cortisone and ACTH therapy.

The physicians report that in 36 per cent (17 instances) of the 47 courses of the drugs administered, cholesterol was elevated to excessive levels. They further observed that the longer the treatment and the larger the dosage, the more pronounced the hypercholesteremia became. Medical researchers believe that a relationship exists between hypercholesteremia and arteriosclerosis.

BLEEDING IN HEART SURGERY

Dr. Samuel A. Thompson, New York, reports how he created an artificial leak-proof valve through which a surgeon could enter the heart without fear of hemorrhage in the November issue of *The Journal of the American Medical Association.* He is associated with the New York Medical College, Flower and Fifth Avenue Hospitals.

The method perfected through research on dogs has been used on two human beings.

To produce an actual working valve, Dr. Thompson sewed two sets of stitches for drawstrings into the tissue of the auricular appendage; one near the tip and the other at the opposite end where it joins the auricle. The tip of the appendage then was cut off to provide an opening.

Dallas to Have New Children's Home

Plans are being made to construct the new Dean Memorial Children's Home in Dallas, which is to be adjacent to the Children's Medical Center, states the Dallas *Times-Herald.* Costing approximately $200,000, the new building will honor the late Dr. John H. Dean of Dallas. The children's home in which Dr. Dean was interested and which he helped by personal contributions originally was known as the Garrett Memorial Home. After Dr. Dean's death in 1929, his widow and son gave the old Dean homestead to the institution.

REACTIONS AND COMPLICATIONS FOLLOWING TRANSFUSION

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THE discovery of the circulation of the blood by Harvey in 1628 made the therapeutic procedure of blood transfusion possible, and in 1665 Lower carried out the first successful transfusion, transferring blood from one dog to another. The first successful transfusions on human beings were done in 1667 by Lower in England and Denis in France. The discovery by Landsteiner in 1900 of the presence of agglutinins in blood serum and the description of the peculiarities of the agglutinating reactions of four groups of blood by Jansky in 1907 and Moss independently in 1910 made transfusion a fairly safe procedure. However, considering the scientific achievements of the early investigators and the improvements made possible by more recent research, far too many transfusion reactions occur even in well conducted transfusion centers. Many of these reactions can be prevented by more careful attention to some of the simple details.

A number of the more common causes for transfusion reactions and complications may be enumerated as follows: (1) blood incompatibility, (2) toxicity of sodium citrate, (3) anaphylactic and allergic reactions, (4) excessive speed of administration, (5) impurity of water and the presence of pyrogens, (6) unclean apparatus, (7) infections (for example, virus diseases, syphilis, malaria), and (8) excessive deposits of hemosiderin.

BLOOD INCOMPATIBILITY

The common factors to be considered in the category of blood incompatibility are errors in typing and cross matching and failure to determine the patient's and donor's status with reference to the Rh factor or other minor agglutinins. Errors in typing may result from the use of low titer typing serums. It is usually a better plan for small laboratories to purchase typing serums of proper titer than to depend upon securing them from available donors. An excellent typing serum is available in the form of a dry powder prepared from the serum of immunized rabbits by the lyophile process. The cell suspension used in typing should never exceed a strength of 2 per cent, as clear-cut readings are not obtainable with a stronger suspension. A serum of satisfactory titer should agglutinate the red cell suspension in a 1 to 20 dilution in less than ten minutes. When the serum is used undiluted, agglutination should be readily noted in from fifteen to thirty seconds. Cross matching should always be carried out after blood types have been determined. Rouleaux formation is often marked in patients with elevated temperatures and may be confusing. It is also noted in patients with increased fibrinogen. Bacteriogenic agglutination (Hübener-Thomsen phenomenon) must be guarded against. Hübener, Thomsen, and Schill and Halberstaedter have shown that dilute suspensions of corpuscles in saline solution kept for some time become panagglutinable, that is, the corpuscles are agglutinated by any human serum without regard to type. According to Friedenreich, this development is due to bacterial contamination of the red cell suspension. Davidsohn and Toharsky have shown that certain Corynebacteria and vibrios may be responsible. It is likely that this reaction is due to the unmasking of a new agglutinogen "T" in red cells through the action of bacterial enzymes.

In addition to types O, A, B, and AB, a number of agglutinable factors in human red cells have been described. The most important of these is the Rh factor discovered by Landsteiner and Wiener[8] in 1940. Other agglutinable factors are M and N (Landsteiner and Levine[7]), factor P (Landsteiner and Levine), factors G and H (Schiff), factor X (Andresen), factor Q (Furuhata and Imamura), and factor E (Sugishita). Of these, the only one of any known importance from the standpoint of transfusion reactions is the Rh factor.

In some quarters it is contended that 90 per cent of all hemolytic transfusion reactions are attributable to the Rh factor, but it is certainly a mistake to attempt to incriminate this factor in all reactions which occur. On the basis of present statistics, 85 per cent of native Americans are Rh-positive and 15 per cent are Rh-negative. Several types of anti-Rh serum have been described, that is, serums anti-Rh_0, anti-Rh′, and anti-Rh″. For practical purposes testing with anti-Rh_0 serum is usually sufficient. In the simplest terms it may be said that an Rh-negative patient might take one transfusion of Rh-positive blood without having a severe reaction. After this transfusion sensitization may occur and a second transfusion with Rh-positive blood may end fatally. It is proper to give an Rh-negative patient only Rh-negative blood. This is especially important in young women who are Rh-negative. Should they receive Rh-positive blood, sensitization may occur and at a later time if they become pregnant and harbor an Rh-positive fetus, complications may occur.

Read before the Section on Clinical Pathology, State Medical Association of Texas, Annual Session, Fort Worth, May 3, 1950.

The Rh factor is of extreme importance in obstetrics. It is said that approximately 10 per cent of marriages are between Rh-negative women and Rh-positive men. Accordingly, a fairly high percentage of sensitized mothers might be expected since the fetuses they carry in utero may inherit Rh-positive blood from their fathers. This, however, is not the case since erythroblastosis fetalis occurs only once in about 400 pregnancies. This figure may be accounted for by the fact that many Rh-negative mothers who give birth to Rh-positive babies never become sensitized to Rh-positive blood during the period of gestation. A number of theories have been advanced in explanation of this peculiar condition. The theory offered by Levine seems the most plausible. He has suggested that defects in the placenta permit an exchange of red cells between the fetal and maternal circulations. The Rh-positive cells of the fetus thereby sensitize the Rh-negative mother. In the normally formed placenta, showing a properly developed Nitabusch's membrane, it may be assumed that maternal sensitization could not occur.

It is of considerable importance to mention the cold hemagglutinin of primary atypical pneumonia as a cause of transfusion reaction. While this agglutinin reacts most actively at ice-box temperature, the possibility must be borne in mind, as pointed out by Dameshek, that it may be responsible for intravascular hemagglutination and hemolysis with the appearance of acute hemolytic anemia and hemoglobinuria. The same author also has reported the presence of a potent hemagglutinin in some cases of other infectious diseases following the administration of sulfadiazine and sulfathiazole. The possibility of reactions, therefore, must be borne in mind in cases of primary atypical pneumonia or in those cases in which both sulfonamide drugs and transfusions of type O blood are given.

Reactions may follow the use of blood from so-called "universal type O donors" when the titer of the donor's serum for the recipient's cells shows agglutination higher than 1 to 16. However, such blood may be rendered safe by the addition of group specific substances A and B of Witebsky. These substances are complex carbohydrates and have been isolated from pepsin, mucin, peptone, and gastric juice. In practice a 1 to 1,000 dilution of each specific substance is prepared and 25 cc. of the stock solution of A and 10 cc. of B are added to each 500 cc. of type O blood. Less of group specific substance B is added because the anti-B factor in O blood is usually much weaker than the anti-A factor. The addition of group specific substances A and B of Witebsky, therefore, makes safe the use of universal donor type O blood in transfusion.

SODIUM CITRATE TOXICITY

Many attempts were made by early workers to find an ideal anticoagulant. In 1667 Major advocated the use of ammonium sulfate for this purpose; in 1860 Neudorfer suggested sodium bicarbonate; a few years later Hicks recommended sodium phosphate; and in 1892 Landois recommended hirudin. A number of other anticoagulants which proved toxic or ineffective were recommended; they included ammonium oxalate, sodium oxalate, arsphenamine, sodium sulfate, sodium iodide, magnesium sulfate, and transfusol (polyacetylene-dioxysulfonate).

Sodium citrate as an anticoagulant was first used by Hustin[6] in 1914. It was used in World Wâr I and also extensively in World War II, not only as an anticoagulant for whole blood transfusions but also in the processing of blood plasma. Sodium citrate has proved to be the best anticoagulant available. Soon, however, certain objections to its use were advanced. Drinker and Brittingham[3] and later Unger[19] suggested the possibility that citrate might be responsible for increased fragility of red cells, reduction of the strength of complement, and alteration of the opsonic index and phagocytic power of leukocytes. Other investigators do not agree with these conclusions. There is no question about the toxicity of sodium citrate when it is used in too large a quantity, and drug idiosyncrasy may be of importance even when the amount is not excessive. Lewisohn on the basis of experimental work with dogs has expressed the belief that 15 Gm. of sodium citrate may be considered as a fatal dose for a man weighing 125 pounds. He has recommended 5 Gm. of sodium citrate as a maximum in blood transfusions. The toxicity of a citrate solution may well depend also upon the speed of administration, as has been shown by Salant and Wise.[15] Tetany may be produced in man with the use of an otherwise safe dose of 6 Gm. if the transfusion is given too rapidly.

Experimental work recently carried out by Bruneau and Graham revealed that exsanguinated dogs survive for a much longer period if blood loss is replaced with heparinized blood instead of citrated blood. The difference, they thought, is due to the toxic action of large amounts of sodium citrate, especially if given over a short period of time. Similar studies were carried out by Ivy and others with both citrated plasma and citrated blood. Their experiments, likewise, showed the danger of administering large amounts of sodium citrate. When sodium citrate is added to blood, a complex salt is formed by the interaction of citrate with calcium. Calcium ions are necessary for the clotting of blood, and when they are combined with citrate, normal clot formation cannot take place. Tetany following excessive use of citrate may be explained in a similar manner.

The use of heparin as an anticoagulant has gained wide popularity in Europe, especially in Sweden.

Hedenius recommended the addition of 0.4 cc. of a 5 per cent solution of heparin to 500 cc. of blood. There are arguments in favor of, and also against the use of heparin, as shown by Bruneau and Graham. The increase of the clotting time in massive transfusions may be so great as to make the use of heparinized blood dangerous. On the other hand, heparinized blood apparently will maintain the blood pressure much better than citrated blood, a point important in the treatment of shock.

Another method of using heparin in transfusion is by directly heparinizing the donor. This is accomplished by administering intravenously 1 mg. of heparin per kilogram of body weight, or about 70 mg. Seven minutes later blood is withdrawn from the donor. Such a method is certainly not without danger to patient and donor alike. The use of heparin has been thoroughly reviewed and studied by Mason.[10]

ALLERGY AND ANAPHYLAXIS

Allergic reactions following transfusion frequently appear. The mildest form is that in which varying degrees of urticaria are noted. In the more severe form there may be spasm of the smooth muscles followed by involuntary urination and defecation, bronchial spasm, and difficulty in breathing. Urticarial reactions of a mild type are said to occur in about 1 per cent of recipients. This percentage coincides closely with the figures of Hoxworth and Skinner, who reported an incidence of 1.8 per cent.

In the anaphylactic form of reaction the patient is highly sensitized and death may follow quickly the introduction of only a small amount of blood. The pathologic changes in the tissues of the body in anaphylactic shock are described in detail by Ziskind and Schattenberg. It is not unlikely that many of the severe reactions attributed by early workers to anaphylaxis were in reality due to the Rh factor.

Reactions due to the hypersensitive state have been explained in a variety of ways. It is possible that precipitins and agglutinins may be produced in a recipient after transfusion. Should the recipient receive blood a second or third time from the same donor following a sufficient period to allow sensitization, that is, ten days or more, a reaction is likely to occur. Incompatibility of leukocytes, the so-called "hemoclastic crisis," or digestive leukocytosis in donors whose blood is used after a meal has been blamed for some reactions by Doan. Experimentally, it has been shown by Mason and others that reactions occurred most commonly in dogs when the donors were fed a meal containing fat. They observed no reactions when both donor and recipient were fed. Numerous reactions due to using nonfasting donors are described in the literature. As was so well pointed out by Price,[12] donor's diet as a possible cause of transfusion reactions should receive careful consideration.

Occasionally a passive transfer to the recipient of hypersensitivity to a food or other foreign substance may occur. Allergy to horse serum may be transferred to a patient receiving blood from a sensitized donor. A case often cited is that of Holder and Diefenbach[5] wherein a recipient became sensitive to strawberries after he had received blood from a sensitized donor. A passive allergic state of this type may last for a variable period, usually from several weeks to three months.

SPEED OF ADMINISTRATION

Excessive speed of transfusion overburdens the right side of the heart and increases the venous pressure. In patients in severe shock in whom there is no cardiac impairment as much as 500 cc. of blood can be given within a period of ten minutes. In recipients in whom cardiac weakness is suspected an excessive rate of administration is not the only danger. As pointed out by Strumia, speed is an important factor in determining transfusion reactions resulting from chemical substances in the transfused fluid. A low concentration of pyrogens or other toxic substances may produce no reaction if the solution is given slowly, but if the rate of administration is increased, the concentration of pyrogens may be raised rapidly and may result in a reaction. On the other hand, there are those who believe that if pyrogens or other toxic materials are introduced in any form of intravenous therapy, reactions will occur regardless of the speed of administration. Milbert has not seen what other workers described as "speed shock" even in unusually rapid rates of injection. He has expressed the belief that fluids can be given as rapidly as necessary when needed to replenish sudden loss of blood or can be given in shock without danger of reaction. When rapid administration is not indicated, as in giving nutrient materials, the slower drip method is advocated.

In an exhaustive study on the velocity factor in blood transfusion, Rothwell and Crocker[14] failed to observe the phenomenon of speed shock. They have stated that if the blood is compatible and untreated by extraneous chemicals, it may be given to the recipient by their technique at a rate of 1 cc. per second without the rate of transfusion being responsible for any reaction. I use the Drummond apparatus, which keeps the blood in contact with the normal vascular endothelium for as short a period as possible and thereby lessens the possibility of reactions.

From experience gained in World War II, I believe that no set rule can be laid down as to the amount of fluid which should be transfused except that enough must be given to raise the blood pressure to 100 mm. of mercury and to maintain it at that level. In one

patient 1 pint may be sufficient; in another 6 pints may be required. I believe that in a case of shock 2 pints may safely be given in fifteen minutes and an additional quantity may then be administered at a slower rate. The amount given should be determined by blood pressure readings. In a patient not suffering from shock a quantity of 2 or 3 pints given at the rate of 40 drops per minute should not be exceeded. The quantity of blood and the number of transfusions to be given may also be determined, of course, by the red cell count and the hemoglobin readings. Ordinarily a transfusion of 1 pint of whole blood will raise the recipient's red blood cell count by 500,000 per cubic millimeter and the hemoglobin from 8 to 12 percentage points.

PYROGENS

Certain substances termed "pyrogens" which are formed in water as a result of bacterial growth and metabolism may act as important and powerful febrile agents. Seibert[17] in 1923 demonstrated conclusively that certain bacteria can flourish in distilled water. If such water is not properly protected, bacterial contamination sooner or later occurs. The pyrogenic substances produced are thermostable and pass through the finest filters.

In an extensive study supported by well-controlled experimental work, Seibert has shown that 50 per cent of the distilled waters from different laboratories produced febrile reactions when injected intravenously into rabbits. She stated that these toxic substances are formed by specific bacteria and can be removed by distillation. These bacteria are nonchromogenic and may be divided into four groups according to reactions produced by their extracts. The toxic pyrogenic substances are readily destroyed by heat in the presence of either acid or alkali, but not by heat alone. When from 80 to 100 cc. of saline solution made with pyrogenic water were injected intravenously in a man, a febrile reaction was noted. No reaction was noted when freshly distilled water was used.

Not only may pyrogens be produced in water through contamination and bacterial growth, but similar pyrogenic production has also been reported in serums. Bullowa called attention to the frequent occurrences of chills and fever after intravenous serum therapy. Felton[4] suggested that such reactions following serum injections may be of bacterial origin since it is so difficult to concentrate serum without some degree of bacterial contamination and growth. Co Tui and Schrift[2] have shown experimentally by intravenous injections in dogs that some common bacteria growing in normal serums may produce a pyrogenic symptom complex consisting of chills, fever, leukopenia, and gastrointestinal disturbances.

There is no doubt that many transfusion reactions can be prevented by the use of pyrogen-free distilled water. Freshly distilled water should be used in preparing intravenous solutions and also in washing and rinsing the apparatus and tubing. The importance of this simple precaution has been well pointed out by Walter. When there is doubt about the purity of distilled water, a simple test for determining the presence of pyrogens may be carried out. This test consists of heating 100 cc. of the water to be tested to 100 C. then adding 10 cc. of 10 per cent sulfuric acid and 0.1 cc. of potassium permanganate. If pyrogens are present, the purple color will be lost after the mixture has boiled for ten minutes.

UNCLEAN APPARATUS

The importance of scrupulous cleanliness and care in the preparation of apparatus used in transfusions cannot be overemphasized. Too often this matter is left in the hands of poorly trained, poorly educated assistants. Fragments of blood may be allowed to adhere to the wall of the tubing used in transfusion. Sterilization by means of the autoclave splits this protein material into fractions which may produce severe reactions when they are washed into the veins of the patient with the next transfusion.

Even though every precaution has been taken to assure proper cleanliness of the rubber tubing, reactions from this source still may occur because of peculiarities in the chemical composition of the rubber tubing itself. This possibility is pointed out in a lengthy article by Busman,[1] who has noted that certain types of pure gum tubing when new seem to contain toxic substances which are responsible for some transfusion reactions. The toxic substance disappears from the tubing after constant use, and the toxic agent can be entirely removed from the tubing by soaking it in a normal sodium hydroxide solution for six hours.

The transfusion service in many institutions has found it advisable to discontinue the use of rubber tubing and substitute tubing made from some of the more chemically inert plastic materials. By such a procedure the percentage of transfusion reactions has been decreased considerably. One of the most desirable types of tubing is viscose tubing which is inexpensive and may be discarded after being used once.

TRANSMISSION OF INFECTION

A number of infectious diseases may be transmitted by blood transfusions, the more common ones being syphilis and malaria. Less common are measles, influenza, relapsing fever, smallpox, and certain virus diseases.

Of these diseases, the most common and most serious is syphilis. When syphilitic infection is transmitted, it is usually in the early preserologic stages. Classical clinical signs in the donor may be totally

absent. It is well recognized that the various laboratory tests for syphilis are negative in the early, and again in the late stages in a fair percentage of cases. Even in the secondary stage, in which a very high percentage of positive Wassermann reactions may be expected, a certain number varying from 1 to 4 per cent may show false negative results. McCluskie[9] and Post[11] have cited instances of the transmission of syphilis in the prechancrous and preserologic stage of the disease. Since the presence of syphilitic infection in the donor cannot always be determined, attempts have been made to sterilize the blood after it has been drawn by the addition of treponemicidal drugs. Salkind[16] has shown that the transmission of syphilis can be prevented in experimental animals by the addition of Chinini bimuriatici in 1 to 1,000 solution to the preserved blood. Other investigators have added arsphenamine, neoarsphenamine, acriflavine, and Mapharsen.

Transmission of syphilis may be prevented by refrigerating citrated blood before use. It has been shown by Turner and Diseker that a storage period of four days is usually sufficient to destroy Treponema pallidum in citrated blood. In blood banks where an ample reserve of citrated blood is kept in cold storage, the plan of not releasing it before the fourth day may prevent the transmission of this disease. By following this procedure, the addition of treponemicidal drugs is unnecessary.

The transmission of infectious agents by means of yellow fever vaccine or blood plasma has attracted much interest. An extensive and important investigation was carried out by Turner and co-workers[18] at Camp Polk, La., of acute hepatitis occurring in soldiers after inoculation with yellow fever vaccine. The disease was indistinguishable clinically from infectious hepatitis or catarrhal jaundice. They concluded that the disease was not yellow fever. However, no attempt apparently had been made to isolate the etiologic agent.

A disease entity which has been recognized with increasing frequency during the past years is hepatitis following whole blood or plasma transfusions. Numerous cases have been reported following the inoculation of soldiers with human serum employed as a vehicle for yellow fever virus. The etiology of this disease has not been definitely proved, but experimental work, both in this country and in England, suggests that the etiologic agent is a virus. Rappaport[13] stated, "Hepatitis has been produced experimentally in human volunteers by parenteral injection, by feeding, and by nasal inoculations of material containing the infective agent. Apparently the virus maintains its virulence for a long period of time in storage in the dried state."

EXCESSIVE IRON DEPOSITS

In certain diseases or special types of anemia it may be necessary to administer whole blood by transfusion over long periods of time. This is especially true of patients with aplastic anemia, anemia in myeloid and lymphoid leukemia, anemia following benzol therapy, anemia due to poisoning with radioactive substances, or anemia following exposure to roentgen ray and radium. In patients with leukemia or aplastic anemia, life has been prolonged by repeated transfusions of blood. Harrison reported a case of aplastic anemia in which 105 transfusions were given over a period of four years.

In cases of repeated transfusion the donor's red cells are destroyed in the recipient after a variable period, usually in from three to four months. When the patient's red cell count again drops to a low level, another transfusion is given. This procedure may be repeated again and again. With the destruction of red cells, hemoglobin is liberated and broken down and simple iron compounds and an iron-free bilirubin are formed. The iron derived from the hemoglobin is stored mainly in the spleen and liver to be used again when more hemoglobin is needed. When excess hemoglobin is introduced, as in oft-repeated blood transfusions, there may be an excessive deposition of iron in the organs of the body and especially in the cells of the reticulo-endothelial system. There may then be noted enlargement of the liver and spleen and pigmentation of the skin and conjunctivas. The parenchymatous organs of the body readily give the Prussian blue reaction for iron. I have found this reaction at autopsy in a number of subjects in whom repeated blood transfusions had been given over a long period of time. The importance of such iron deposits as a cause for interference with the normal functioning of vital internal organs must be borne in mind.

SUMMARY

Transfusion reactions may be attributed to many causes, among which is improper care in preparation of the transfusion apparatus and negligence in the preparation of solutions for use in transfusions. The toxicity and effectiveness of various anticoagulants in comparison to heparin, as well as the importance of the Rh factor as a cause for reactions, are discussed. The use of so-called "type O conditioned blood" prepared by adding Witebsky substance A and B, which reduces the agglutinin titer, lessens the danger of reactions. There is danger of transmission of such diseases as syphilis, malaria, and certain virus diseases through blood transfusion. It is shown that hepatitis has followed the use of yellow fever vaccine when prepared in a vehicle of human plasma. A similar condition followed the use of blood plasma when given to combat shock. One of the dangers of too frequent blood transfusions is the building up of ex-

cessive iron deposits in the tissues and organs of the body which may interfere with their proper functioning.

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ABSTRACT OF DISCUSSION

DR. W. N. POWELL, Temple: Those of us who have to do with the operation of blood banks are much concerned with the reactions and complications which may follow blood transfusions. This responsibility involves the selection of blood donors, the typing and cross matching of donor and recipient, the collection and storing of blood, and its proper administration to the patient.

No matter how carefully one selects donors, it is not possible to exclude all who might conceivably transmit disease to the patient. The rejection of persons with fever and infections of the upper respiratory tract helps guard against the transmission of some of the common contagious diseases. Routine serologic testing and the refrigeration of blood for three or four days virtually exclude the possibility of transmitting syphilis. Malaria may be an important problem in regions in which it is endemic. Even the examination of thick blood smears for plasmodia and the storing of blood for considerable periods do not entirely insure against its transmission. Various suggestions, from rejection as donors of all persons who have ever had malaria to exclusion of all who have had active disease within two years, have been made. It would appear that a compromise between these extremes, perhaps freedom from evidences of active malaria for five years without suppressive therapy during the period, would be a reasonable rule for most transfusion services to follow. Homologous serum jaundice is a live problem at present. No matter how much care is used in screening donors, those who have had nonicteric infections may not be detected and occasionally infection of recipients will occur.

The use of commercial bottles and disposable tubing kits has simplified the safe collection and storage of blood and has reduced the incidence of pyrogenic reactions. Careful investigation of alleged pyrogenic reactions not infrequently indicates that the chill and fever are due to the primary disease and not to the transfusion.

Even in the most efficient blood transfusion services, sooner or later hemolytic reactions seem inevitably to occur. In my experience these more often have been due to errors in the administration of the blood than to laboratory errors in typing and cross matching. Failure to read labels and transfusion sheets carefully, confusion of bottles, and similar errors are all too often the cause of these distressing occurrences. In the conscious patient, the slow administration of the first 100 cc. of the transfusion material usually permits recognition of reactions soon enough to prevent serious consequences. Unfortunately, this last safeguard is abolished when the transfusion is given to the unconscious patient on the operating table. In the latter instance it may not be apparent that a reaction has occurred until anuria is noted. When a reaction occurs during or following a transfusion, the blood and urine of the patient should be examined for free hemoglobin and proper checks made for possible incompatibilities. When it is established that a hemolytic reaction has occurred, the patient should be treated according to the principles laid down by Muirhead, Hill, and their associates. Under such a regimen a favorable outcome may be anticipated in a high proportion of cases.

Anticoagulant Drugs in Heart Disease

To help reduce deaths and disability resulting from heart disease, more general use of anticoagulant drugs in the treatment of coronary thrombosis is recommended by Dr. Irving S. Wright, professor of clinical medicine, Cornell University, New York. He bases his recommendations on recent controlled experiments with heart patients as described in the Clinical Progress section of the December issue of *Circulation*, the journal of the American Heart Association. He suggests further that other drugs recommended for therapy in coronary heart disease be subjected to similarly controlled studies before they are accepted by the medical profession as being of established value.

Dr. Wright outlines the established therapy as including rest, sedatives, alcohol, oxygen, and transfusions as well as the administration of anticoagulant drugs. Described are heparin and Dicumarol. The newer anticoagulant drug, Tromexan, is mentioned in the treatment of coronary thrombosis which kills at least 200,000 persons a year in the United States.

Trained employees are more valuable to an organization than new employees, and periodic health surveys may prevent many physical disasters and prolong the working life and capacity of faithful workers.—Edward I. Salisbury, M. D., F.A.C.S., New York State J. Med., September 15, 1950.

MICROTECHNIQUES OF BLOOD CHEMICAL ANALYSIS IN PEDIATRICS

Indications and Methods

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CHEMICAL analyses of the blood are as frequently indicated in diseases of infants and children as in those of adults. Unfortunately, often the pediatrician is forced to forego the use of these diagnostic adjuncts since most laboratories routinely request blood samples of such magnitude as to contraindicate their usage in infants and small children.

It is the purpose of this paper to point out: (1) why laboratories request relatively large blood samples, (2) why the requests of such volumes of blood are incongruous to the practice of good pediatrics, and (3) how adequate laboratory studies of the blood may be made without undue exsanguination of pediatric patients.

REQUESTS FOR LARGE SAMPLES

In any procedure in which spot sampling is done and the results indicate the concentration in the whole, it is extremely desirable in the interest of accuracy to use as large a part of the whole for sampling as possible. Also, the concentration of most of the chemical substances of physiologic significance are relatively small per unit volume of blood; in fact, their concentrations are so small that they frequently are reported in parts per hundred thousand parts (milligrams per hundred cubic centimeters). Thus, to give measurable amounts of one particular chemical constituent of the blood a relatively large sample is indicated.

In consideration of these facts, among others, early investigators devised techniques that required volumes of blood of the order of 10 ml. or more for most determinations. As soon as it became apparent to most manufacturers of laboratory appliances that blood chemistry procedures were to be routine, equipment was made to meet the specifications of the original investigators. Thus, because of valid reasons for using relatively large spot samples and the proved degree of accuracy expected by following the techniques of original investigators with standard equipment, most clinical laboratories have followed routine procedures that require relatively large blood samples. Among numerous other reasons for such a practice is the possibility for repeating a test on a sample in case of accident or a questioned result.

From the Department of Clinical Pathology, University of Texas Medical Branch.

Read before the Section on Pediatrics, State Medical Association of Texas, Annual Session, Fort Worth, May 2, 1950.

LESS BLOOD FOR PEDIATRICS

The existence of scores of blood banks throughout the country is mute evidence to the general belief that ill patients need not only all of their own blood for defense but frequently supplements. Therefore, removal of any blood from a patient is contraindicated unless the injury sustained is more than compensated by the information obtained.

Adult patients sustain but little insult by the removal of 10 ml. of blood. However, I do not believe that the average physician gives much thought to the significance of the loss of 10 ml. of blood from a child.

Although estimates of the blood volume vary considerably, dependent to some extent on the method of measurement, most investigators agree that the volume is proportional to body weight, about 80 to 90 ml. of blood per kilogram of body weight. Thus, the average 150 pound man has a blood volume of more than 5,000 ml. and the removal of 10 ml. of blood represents a loss of only one five-hundredth of his blood. In the average 4 year old child, however, such an amount represents one one-hundredth of his blood volume; while in an infant the removal of 10 ml. of blood represents the loss of one-thirtieth of the defenses inherent in the blood. If the physician merely would consider that the loss of 10 ml. of blood in an infant is analogous to the loss of approximately 165 ml. in the average adult, such procedures would be performed infrequently. When the harmful effects of excessive blood loss in the child or infant are weighed against the obvious technical disadvantages of dealing with small spot samples, it seems evident that macromethods of chemical analyses of blood should be abandoned in pediatric patients.

DECREASING REQUIRED BLOOD

Maximum exploitation of the diagnostic features of blood chemistry with stringent conservation of the blood of children is the dual responsibility of the pediatrician and the clinical pathologist. The pediatrician must recognize that blood chemistry studies, unlike urinalyses and blood counts, do not belong in the realm of routine tests. Such tests should be performed only to verify clinical impressions obtained from critical examination of the history and physical findings. An obvious imperative corollary is that the

pediatrician be familiar with the indications for the usual tests.

Through familiarity with the usual blood tests the pediatrician can avoid requests for blood chemistry determinations which are a duplication of other tests or in which the suspected organic dysfunction can be more easily ascertained by other means. Examples are as follows:

1. Blood nonprotein nitrogen and blood urea determinations should not be requested on the same patient.

2. A nonprotein nitrogen determination should not be used as evidence of kidney dysfunction unless the more sensitive dilution and concentration tests are positive. Of course the latter tests may be impossible in small children because of difficulties in the collection of urine.

3. Except when gout is suspected, which is unlikely in pediatrics, a blood uric acid determination should not be requested when the nonprotein nitrogen level is elevated. Uric acid is one of the first nitrogenous substances retained by the kidney and therefore represents superfluous information.

4. A blood creatinine determination should seldom be requested unless the nonprotein nitrogen level is at least 50 mg. per 100 cc. of blood, as results of the determination will not be positive until the nonprotein nitrogen is of a greater magnitude.

Knowledge of the different types of examinations which may be done on a protein-free filtrate, blood serum, or an oxalated whole blood specimen will permit the pediatrician to obtain the maximum amount of diagnostic information from a single blood specimen. It also will prevent such profligacy as requesting a blood sugar determination one day and a nonprotein nitrogen determination the next, for a 1 ml. sample of blood forms more than enough protein-free filtrate to make both tests. When such grouping is requested, the tests desired should be listed in the order of their importance to the physician to insure the performance of at least the paramount tests before the specimen is exhausted.

The clinical pathologist, having experimental proof that as the size of the spot sample is decreased accuracy decreases and labor and cost increase, feels justified in demanding large blood samples. However, the fundamental premise motivating the request for any laboratory procedure is the desire to improve the state of health of the patient; one has but to recall the importance of blood in bodily defenses and to visualize the relative paucity of blood possessed by pediatric patients to be struck by the incongruity of such macroprocedures. Therefore, the pathologist must sacrifice some convenience of performance, increase his labor, obtain some new equipment, and perhaps even endanger the desired degree of accuracy to give diagnostic aid without causing excessive insult to the patient.

There are at least three types of procedures which may be instituted in the interest of pediatric patients by the clinical pathologist:

1. Multiple determinations may be made on a single sample. This type of procedure is wasteful of personnel and equipment and is not feasible in a busy laboratory except as a special concession to pediatric patients.

2. Many standard techniques which are extremely wasteful of blood can be rather easily converted to semi-microtechniques. The following are a few examples of tests wherein an excessive amount of blood is routinely used for increased ease and accuracy of manipulation:

   a. *Ascorbic acid.*—Two ml. of plasma are diluted to 10 ml. with water and metaphosphoric acid, and only 2 ml. are used.
   b. *Alkaline phosphatase.*—One ml. of serum is diluted to 20 ml. and only 6 ml. are used.
   c. *Albumin determination.*—Five-tenths ml. of serum is diluted to 10 ml. with sodium sulfite. This is filtered and only 1 ml. of filtrate is used.
   d. *Total proteins.*—One ml. of plasma is diluted to 50 ml. Of this suspension 3 ml. are used in the Biuret method or if the digestion method is used, only 1 ml. is needed.
   e. *Protein-free filtrates* (for nonprotein nitrogen glucose, urea, creatinine, and so forth).—Two ml. of blood are diluted to 20 ml. Of the 10 ml. or more of filtrate obtained only 2 ml. are used for a sugar determination and 5 ml. for a nonprotein nitrogen determination.

It is apparent from the above examples that considerably less blood could be required by merely changing the equipment and making dilutions at the same ratio on a smaller scale. It is to be remembered, however, that in dealing with small amounts of blood, the possibilities of error are enhanced and any error is magnified in the result.

3. True microtechniques may be established for certain tests, especially those which may be serially requested:

   a. The carbon dioxide capacity can be accurately determined on 0.2 ml. of plasma in the manometric Van Slyke apparatus.
   b. Flame photometry can accurately determine sodium, potassium, calcium, and other anions in 1 ml. of serum. Microchemical analyses can be developed for each of the anions if a flame photometer is not available.

SUMMARY AND CONCLUSIONS

Large amounts of blood for blood chemistry studies in pediatrics are contraindicated. The pediatrician can lessen the insult concomitant with blood chemistry studies by judiciously requesting only pertinent tests. Standard laboratory procedures can be modified whereby they become semi-microtechniques. The pediatrician and clinical pathologist can develop measures to derive the maximum amount of diagnostic information from a given blood sample.

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ABSTRACT OF DISCUSSION

DR. MATHEW D. BURNETT, Houston: The need for improvement in the chemical techniques of laboratory studies on infants and children cannot be overemphasized. Dr. Baird's review of microtechniques questions the widely-held opinion that unfortunately such analyses in the past usually have been limited to those laboratories forming integral parts of pediatric hospitals. It seems that some fallacy lies in the assumption that true microtechniques, even within the limits of error of the methods, can be done with accuracy in general hospitals. Evidently it has not proved economically practical in these latter institutions to maintain and allocate the additional equipment, special technicians, and space required for microchemistry, when the bulk of the chemistry department is geared for large blood samples.

The most important requirement of a well-organized microchemistry laboratory in a general or a pediatric hospital is a full-time pathologist who not only is efficient and experienced but who is ever cognizant of the narrow margin of error in microchemical techniques. Conscientious and frequent checks on his techniques, technicians, and equipment are essential to maintain high standards of accuracy.

Dr. Baird has warned attending physicians against the withdrawal of large amounts of blood for analyses. A single specimen hardly ever will cause untoward symptoms, but occasionally cumulative bleeding because of repeated sampling is unavoidable. This possible danger could better be foreseen if the time and amount of withdrawals of blood were recorded on the patient's chart. Replacement by transfusion might be necessary at times and such a record would be valuable in calling attention to the need.

There are innumerable times when the pediatrician is grateful to have microchemistry available for the study of his patient, but hardly ever is it so valuable as in the premature infant. "Prematurity" is the most common cause of death in the neonatal period. Yet it must be admitted that premature infants probably receive less study of their blood chemistry than patients in any other age group because so often their general physical condition contraindicates withdrawal of large specimens of blood. It is reasonable to assume that the mortality rate of premature infants could be reduced significantly if just the electrolyte balance could be determined repeatedly when indicated without producing anemia by exsanguination.

The practical conclusion seems to be that the clinician must cooperate with the pathologist in establishing the laboratory method of blood chemical analyses best adapted to the individual institution and most conservative in the amount of blood sampling required for infants and children.

NEUROLOGIC JOURNAL STARTED

The first issue of *Neurology*, a publication to be sponsored by the American Academy of Neurology, is being mailed in January, 1951. The bimonthly will embrace every aspect of clinical neurology and will be designed for both the specialist and general practitioner.

The editor-in-chief will be Dr. Russell N. DeJong, professor and chairman of the Department of Neurology, University of Michigan. The journal will be published by Lancet Publications, Inc., Minneapolis, publishers of the *Journal-Lancet, Modern Medicine,* and *Geriatrics.*

TEXAS CANCER CLINICS RECEIVE AID

Two Texas medical clinics recently have received financial support from the Texas Division of the American Cancer Society. Of twenty-eight full and part-time tumor clinics in the state, Texas Cancer Crusade funds now aid twelve.

A newly established clinic at Waco, which will serve cancer patients of several Central Texas counties, will receive $3,000 from the Society. In Houston funds of $1,200 will help the surgical follow-up service at Hermann Hospital.

SPEECH TRAINING FOR CANCER VICTIMS

An appropriation of $5,500 has been authorized by the executive committee of the Texas Division of the American Cancer Society to aid a program of speech training for victims of cancer who have lost the power of speech by the removal of the larynx.

Since last April a post-laryngectomy speech-training class has been operated on a trial basis at the M. D. Anderson Hospital for Cancer Research, Houston. Of nine students, none of whom could utter a word when the classes began, every one has regained his speech. The class is the only one of its kind in this geographic section and one of only two or possibly three in the country.

With the aid of the Texas Cancer Crusade funds the course will be able to help applicants from all over Texas. It is estimated that there are approximately 174 new cases of cancer of the larynx each year in Texas and that almost 500 such patients are now living in the state.

SERUM SODIUM AND POTASSIUM

Studies With the Beckman Spectrophotometer and Review of the Literature

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DURING the past fifteen years tremendous advances have been made in the knowledge of the distribution of body water and the salts dissolved in it. The continuous flow of water in and out of the three body compartments, intracellular, interstitial, and vascular, so necessary for the transport of nutrient material and wastes is maintained by physical forces which modern research in the fields of physical and biologic chemistry have revealed. The failure of flow and the depletion or retention of fluids in one or another compartment due to imbalance of dissolved salts results in death, often without visible changes in morphology. The internal chemistry of the cell involved in vital processes also requires that the dissolved salts be present in quantities within certain limits. Therefore it is essential that students of disease understand the "chemical anatomy and pathology of the extracellular fluid," as Gamble[13] puts it, as thoroughly as they understand normal and abnormal morphology.

Serum studies are not enough, for they reveal only serum concentration.[1] Urine studies will reveal that which the kidney is withholding or excreting. To these observations must be added a full knowledge of the patient's history, signs, and symptoms. A physician familiar with the basic physiology then may reason the imbalance which is present or most likely to occur.

BASIC PHYSIOLOGY

Sodium

Sodium is the chief cation of the extracellular (interstitial and intravascular) fluid. The osmotic value of extracellular fluid is composed for all practical purposes of the sum of the concentrations of its ions. The total concentration of ions measured in milliosmols is determined by the sum of the cation values. Because the HCO'_3 ion is adjustable and changes in other anions will be offset by changes in the HCO'_3 ion and so will not bring about change in the total ionic concentration, nearly all of the cation concentration is sodium. Therefore the stability of the osmotic value of extracellular fluid depends much upon the efficiency of the kidney in the control of sodium excretion and retention. The experiments of Darrow and Yannet show the action of sodium in a fascinating manner. If salt is introduced into the extracellular fluid, water passes from the intracellular compartment into the extracellular compartment. The volume of the extracellular compartment rises, and the volume of the intracellular compartment falls. The reverse occurs if salt is withdrawn. In either instance, osmotic equilibrium is approached. The kidney if normal then restores the normal ionic concentration, and volume dimensions assume their usual values.

This whole mechanism depends, as stated above, on normally functioning kidneys and also available water or sodium. Serum concentration will not reveal necessarily either interstitial or intracellular concentrations of water. Normally acting kidneys will, within certain limits, withhold water or salt. Renal salt economy is more efficient than water economy. Salt depletion may be revealed easily by the Fantus test.[10]

In addition to its role in the maintenance of water balance and osmotic equilibrium, sodium is also concerned with acid-base balance and maintenance of normal muscle irritability. The presence of sodium is necessary for normal cardiac muscle contraction.

Salt Depletion.—Salt depletion may result from a number of causes:

1. Abnormal salt losses. Abnormal salt losses are the chief cause of salt depletion. Reports of various investigators point to the astonishing amounts of salt which may be lost in twenty-four hours: sweat 1 to 40 Gm., vomitus 40 Gm., gastric juice (suction) 18 Gm., intestinal juice (suction and diarrhea) 20 to 50 Gm., and bile or pancreatic fistulas 4 to 30 Gm. Inadequate intake of salt which might well accompany any one of these conditions and excessive hydration which may be enforced in these days of "fluid consciousness" will exaggerate the salt losses.

2. Chronic renal disease. The kidney loses its ability to substitute ammonia for sodium. Further, the tubular epithelium may fail in its ability to absorb sodium.

3. Other causes. The occurrence of salt depletion in diabetes, infectious disease, and adrenal cortical insufficiency is well understood.

In therapy of cardiac disease there is danger in combining salt restriction and mercurial diuresis. Salt depletion results in the excretion of water by the kidneys to bring about extracellular isotonicity. Both

Read before the Section on Clinical Pathology, State Medical Association of Texas, Annual Session, Fort Worth, May 3, 1950.

plasma and interstitial fluid volume fall, and there is a fall of extracellular osmotic pressure evidenced by lassitude, weakness, headache, giddiness, anorexia, nausea, and vomiting. The latter tends to exaggerate still further the salt loss. Cramps and mental confusion occur in severe cases. The patient shows the evidences of dehydration in his general appearance. Blood pressure falls and there is circulatory failure.

Salt Retention.—The chief cause of salt retention is diminished renal blood flow. Diminished renal flow in advanced kidney disease or arterial spasm is understandable. In congestive heart failure the mechanism is debatable, but it is agreed that there is diminished renal blood flow and increased salt absorption from the tubule so that there is diminished salt retention. Retention of sodium does occur with retention of water and a rise of extracellular fluid volume. To establish isotonicity, water is withdrawn from the cells, and though the patient is edematous, he suffers from cellular dehydration. Restriction of water in such cases only serves to exaggerate the imbalance.

Adrenal cortical hormone and the pituitary antidiuretic hormone also play a role in salt retention.

Potassium[3, 5, 17, 30]

Ingested potassium is rapidly absorbed from the gastrointestinal tract and diffuses from the blood stream into the cells of the body, where the largest portion of body potassium exists. Potassium is lost from the body in the sweat and in the stools via the gastrointestinal juices, which are rich in potassium, but more than 90 per cent is lost through the kidneys. Potassium enters the urine by simple glomerular filtration; some is reabsorbed in the tubule by substitution with the ammonium ion, but the rest is excreted. Under certain circumstances, excretion of potassium through the renal tubule can be unequivocally demonstrated. Between 3 and 4 Gm. of potassium are ingested and the same amount excreted each day.

The adrenal cortex, more specifically the steroid hormones elaborated by the zona glomerulosa, seems to act in a general supervisory capacity over the ingestion, distribution, and excretion of potassium. It regulates the amount of potassium that will be taken up by cells, the distribution between cells and fluid, the relative proportions of sodium and potassium to be found in sweat, and the amounts that will be excreted in the urine and thus governs the resultant concentrations of these electrolytes in the extracellular fluids.

Human serum contains from 4.09 to 5.63 milliequivalents of potassium per liter (16 to 22 mg. per 100 ml.); interstitial fluid, of course, has the same amount. However, cells contain about twenty times this concentration of potassium; red cells hold tremendous amounts, averaging 420 mg. per 100 ml., while about three-fourths of the body's total potassium is in muscle cells. Cellular potassium is only partly ionized, the rest existing in combination with protein and glycogen. However, it is alterations in the concentration of potassium in the extracellular fluids which affect the function of various body organs such as the heart, nerve, and muscle, and produce the clinical syndromes of hyperpotassemia or hypopotassemia. The concentration of potassium in the extracellular fluid compartment is a result of the amount of potassium entering this compartment from the gastrointestinal tract and from cells and the amount leaving the compartment via the urine, sweat, gastrointestinal secretions, diarrhea or vomitus and by entering cells. The concentration can further be altered simply by dehydration or rehydration, in which the total amount of potassium is not changed but in which shrinkage or expansion of the extracellular fluid compartment occurs. Only a major functional disturbance will alter potassium concentration, but when this alteration occurs, the consequences are serious.

Potassium is integrally concerned with the function of the heart in that it increases the diastolic time of the cardiac cycle, and if in high enough concentration will cause cardiac arrest in diastole; insufficient potassium will result in small focal areas of necrosis which are replaced by fibroblasts, with clinical evidences of severe myocardial damage.[6] Potassium is probably necessary for the transmission of the nerve impulse along the neuronic pathways and is also essential in transmission of the impulse across synapses as well as across the myoneural junction. It is also essential for the process of muscular contraction; it has been found to leave the muscle cell on contraction and reenter at rest. In an as yet incompletely explained manner, this element is related to acid-base balance in that hypopotassemia and hyperpotassemia frequently occur with alkalosis and acidosis respectively. Other important roles of potassium involve carbohydrate and protein metabolism. Whenever glucose is being polymerized in the liver to form glycogen, potassium and phosphate are laid down with it in a definite weight-to-weight ratio of potassium to glycogen; when glycogen is broken down and glucose is liberated into the blood stream, potassium is liberated with it. Similarly, when protein is laid down in tissue cells and the nitrogen balance is positive, potassium is deposited with it in a ratio of 1 Gm. of nitrogen to 2.38 milliequivalents of potassium; and when protein catabolism prevails, potassium is liberated from the cells into the extracellular fluids.

HYPOPOTASSEMIA

The signs and symptoms of hypopotassemia are limited to the cardiovascular and skeletal systems and

occur when the serum potassium concentration reaches 2.5 to 3.0 milliequivalents per liter (9.8 mg. per 100 cc.). The most conspicuous feature is generalized muscular weakness (at 3.0 milliequivalents per liter) or flaccid paralysis (at 2.5 milliequivalents per liter); this involves all of the striated musculature of the body, though usually not involving the muscles above the neck. Respiratory difficulty characterized by a rapid, difficult, shallow, gasping type of breathing, accompanied by poor chest expansion and flaring of the alae nasi, occurs as a result of paresis of the voluntary muscles of respiration. Difficulty in swallowing and in speaking may occur because of paralysis of the muscle groups necessary for these functions. Accompanying these paralyses are a loss of deep tendon reflexes and a low electrical excitability of muscle. There is preservation of sensation, and the patient remains alert and his mind clear while in the midst of an attack of hypopotassemia. With reduction of serum potassium to this critical level profound alterations in cardiac muscle physiology occur; these become clinically evident by an increased pulse pressure, a decreased diastolic pressure, a collapsing type of pulse, an increased venous pressure, cardiac dilatation with an increased area of cardiac dullness, systolic murmurs, and cardiac arrhythmias.

Pathognomonic electrocardiographic changes accompany these clinical manifestations (fig. 1). These include most notably negative T waves, an increase in the QT interval due to a prolongation of a T wave, depression of the ST segment and occasionally U waves. The T wave changes are most evident in precordial leads in the third position (CR_3, CF_3, V_3), where T waves are most apt to be widest and clear-cut. The U waves differ from those sometimes seen in the normal electrocardiogram in that they disappear after the administration of adequate potassium. It is thought by some that the wide T wave in this condition is the result of a combination of a T wave and U wave. It is interesting to note that hypocalcemia will also cause a prolongation of the QT interval, but this prolongation is due not to lengthening of the T wave but to an increased ST interval. The electrocardiographic manifestations of hypopotassemia do not appear consistently at specific potassium levels, since it has been noted that cardiographic changes may occur at different levels in patients who have been digitalized; it has further been postulated that hyponatremia, when it coexists with hypopotassemia, may mask the cardiographic changes which occur in the latter condition.[8] In spite of these variabilities some workers regard the electrocardiographic changes as a rather good index of the serum potassium level, of particular use in the treatment of diabetic coma.

Causes of Hypopotassemia

Diabetic Acidosis.[14, 16, 22, 24, 25, 32]—Most physicians are acquainted with the syndrome of flaccid paralysis, respiratory difficulty, and death which occurs in many patients in diabetic coma after they have been vigorously and properly treated for from twelve to twenty-four hours. Recent work has shown this syndrome to be due to a marked lowering of the serum potassium concentration. This hypopotassemia has been found to occur in almost all treated cases of diabetic acidosis, though the lowering does not always reach clinical levels. There are many causes for this derangement in blood chemistry, but the condition can easily be understood by following the natural history of a person with uncontrolled diabetes.

The person with marked hyperglycemia, glycosuria, and polyuria loses considerable amounts of potassium as a result of the large volume of urine excretion. As the person gets sicker and as the acidosis gets more intense, body stores of potassium are further decreased by an excessive loss of potassium in the vomitus plus the fact that there is a decreased potassium intake in the food. This person who is going steadily downhill is in negative nitrogen balance and negative glycogen balance. Thus, as his cellular protein is being broken down and his liver glycogen stores are being depleted, potassium leaves the liver and tissue cells, enters the extracellular fluid, and is made available for excretion via the urine and vomitus, thus offering another important mechanism for body potassium depletion. At this point the patient is going into coma or is in coma and enters the hospital for treatment.

On his admission to the hospital the serum potassium concentration may be either normal or high in spite of the fact that total body potassium stores are unusually depleted. This is because concomitant dehydration with marked diminution of extracellular fluid volume may mask the lack of potassium since both fluid volume and potassium may be proportionately decreased. At this point vigorous treatment with large amounts of parenteral fluids, insulin, glucose, and salt is instituted. Diuresis is reestablished and more potassium is again lost in the urine. As glycogen becomes deposited in the liver and as positive nitrogen and sugar balances are reestablished in the various cells of the body, potassium leaves the already depleted extracellular fluid and is deposited in the liver and body cells along with the glycogen and protein. Finally, hemodilution with large amounts of parenteral potassium-free fluid further decreases the concentration of potassium in the extracellular fluids.

Interesting studies have been made on other minerals in diabetic acidosis. It has been shown that there is a striking fall in the concentration of magnesium after the first twenty-four hours of treatment and that the concentrations of sodium and calcium show little or no changes during the entire treatment period. The

exact role of magnesium has not yet been thoroughly assayed.

Renal Disease.—The normal kidney cannot conserve potassium as it does sodium, even in the presence of hypopotassemia or depleted body potassium stores. As long as urine is formed, potassium will be excreted. Normally potassium is excreted via the glomerulus and some of it is reabsorbed in the tubule, where it is replaced by the ammonium ion as part of the kidney's base conserving function. However, in altered tubular functional states, such as occur in certain forms of nephritis, the kidney loses its ability to substitute ammonium for potassium and sodium and thus excessive amounts of potassium and sodium are lost via the urine.[22] This is sometimes called a "salt-losing nephritis." In other kidney diseases in which there is oliguria or anuria, where renal blood flow and glomerular filtration are noticeably diminished, there is an increase in serum potassium due to diminished excretion. Thus either hypopotassemic or hyperpotassemic states may result from kidney disease, depending upon whether the functional defect is primarily that of tubular reabsorption or glomerular filtration.

One series of cases in which 26 patients with uremia due to assorted[9] renal diseases were studied showed that more than one-half had abnormally high serum potassium values (more than 5.5 milliequivalents per liter); 11 of these patients had values of more than 6.4 milliequivalents, which is in the range where electrocardiographic changes begin to appear. In most of these patients the serum potassium rose as the renal insufficiency and oliguria progressed, and it was shown that oliguria or anuria were prerequisites to the hyperpotassemia which occurs in renal diseases. Four patients in this series demonstrated abnormally low potassium levels. No correlation could be established between the pathologic type of renal disease and the alterations in serum potassium concentration. The dangers of administering potassium via the oral or intravenous route to patients with anuria or oliguria can clearly be visualized.

Studies have also been made upon the electrocardiographic patterns found in uremia.[20] These changes are due to a combination of factors: (1) left ventricular strain, (2) coronary artery disease, (3) diffuse pericarditis, (4) hypocalcemia, and (5) hyperpotassemia. However, a combination of prolonged electrical systole of the hypocalcemic type together with narrow, tall T waves indicative of potassium poisoning is the most characteristic, though not the most commonly found, electrocardiographic feature of uremia; this combination, however, can also occur in Addison's disease. A frequently found combination is that of hyperpotassemia superimposed upon a left ventricular strain pattern, in which the inverted T wave in leads 1 and CF_5 becomes much less deep as a result of the elevated potassium level. It has been postulated that potassium poisoning with cardiac standstill is the immediate cause of death in at least one-fifth of the cases of uremia in men, and thus the electrocardiogram offers a good means of prognosis in these cases.

Diarrheal States.—Numerous reports of the hypopotassemic syndrome occurring together with various diarrheal states have been made. Chinese authors have reported many cases of Pa-Pin (transient paralysis) complicating Asiatic cholera;[18] all of the patients developed paralysis shortly after recovering from cholera, subsequent to the administration of massive amounts of potassium-free parenteral fluids; all responded well to treatment with potassium. There has been reported a case of severe chronic functional diarrhea in which there was marked depletion of body potassium resulting in characteristic electrocardiographic changes and marked asthenia; the patient died during an infusion of intravenous glucose, and myocardial lesions which were highly suggestive of potassium deficit were found at autopsy. It has been shown further that the formerly high mortality rate in infantile diarrhea was due to the marked lowering of serum potassium;[7] since this fact has become known and since the addition of potassium to the therapeutic regime for these infants has become routine, the mortality rate has been decreased from 32 to 6 per cent. Actual paralysis has not been observed in these cases, though loss of muscle tone is a frequent clinical observation. Several cases of sprue have been reported in which the diarrheal state led to low serum potassium levels.[15] The potassium deficiency in all of these instances is due to a poor intake of potassium from malnutrition and the loss of tremendous amounts of potassium in the intestinal fluids, augmented by therapy with large amounts of potassium-free parenteral fluids. Post-surgical upper gastrointestinal fistulas of all types may also lead to a hypopotassemic state because of the loss of large amounts of potassium in the intestinal secretions.

Familial Periodic Paralysis.[29, 31]—Periodic paralysis, which is familial in 80 per cent of the cases, is characterized by attacks of flaccid paralysis of the skeletal musculature with a clinical picture almost identical to the syndrome of hypopotassemia. This rare ailment, which sometimes occurs with thyrotoxicosis, does not become manifest until puberty.

In all likelihood the attacks are due to periods of hypopotassemia though this is doubted by some authors. During an attack most patients show low serum potassium levels; between attacks the potassium levels are normal. It is not known exactly where the potassium goes during an attack; it has been shown that it is not lost in the urine and the prevailing

opinion is that it is deposited in the liver and in tissue cells. This theory is substantiated by the fact that attacks are precipitated by the administration of large amounts of glucose or insulin or both, that attacks come usually in the early morning, eight to twelve hours after a heavy carbohydrate meal, and that attacks will be brought on regularly by 200 Gm. of glucose administered orally. Recent work points to the fact that the basic disorder of this disease probably involves the hexose-phosphate phase of carbohydrate metabolism in the liver and that the basic defect is a disorder of the contractile mechanism of muscle itself, possibly based on a chronic deficit of potassium. Most cases respond promptly to treatment with potassium given orally or intravenously, and a return to normal potassium values has been an almost constant observation as the paralysis subsides.

Some authors doubt the role of hypopotassemia in this disease and base their arguments upon the fact that normal people given adrenalin or glucose show reduction in potassium to low levels, yet no paralysis, that intravenous potassium administration gives no relief in some patients, and that some patients have been observed during attacks in which the potassium concentration was normal.

Intestinal Obstruction.—A high percentage of patients with intestinal obstruction have been found to develop low serum potassium concentration together with electrocardiographic changes thereof and weakness.[2] The causes are many: (1) excessive amounts of potassium are lost in the vomitus; (2) with the installation of therapy, continuous Wangensteen suction causes further loss of potassium; (3) the negative nitrogen balance occurring with protein breakdown in this state causes potassium to leave tissue cells, to enter extracellular fluids, and thus be made available for further loss by the previous routes; (4) administration of parenteral glucose causes deposition of potassium in the liver; (5) the diuresis which is reestablished on treatment causes further loss in the urine of badly needed potassium; and (6) the administration of large amounts of potassium-free intravenous fluids causes dilution of the potassium already present.

In a series of 15 patients with intestinal obstruction and protracted vomiting, all were found to have low potassium concentrations, and these levels decreased still further after therapy with saline and glucose solutions.[2] Varying grades of muscle weakness and asthenia were present in these patients though no real paralysis was noted. The electrocardiogram was found to revert to normal when intravenous potassium was administered, whereas no effect was noted with the administration of intravenous calcium. It is postulated, therefore, that some of the signs and symptoms of intestinal obstruction may be due to hypopotassemia. Interesting electrolyte studies in these cases showed that where high obstruction existed there was a slight decrease in sodium, a decrease in chloride, hypopotassemia, hypocalcemia, and alkalosis, whereas in lower obstructions the same pattern existed except for acidosis and a markedly decreased sodium value. Other diseases accompanied by excessive vomiting have also been mentioned sporadically as causes of hypopotassemia.

Miscellaneous.—Hyperadrenocortical states, due to the specific action of cortical hormones on sodium and potassium metabolism, have been demonstrated to cause signs and symptoms of hypopotassemia. This set of circumstances has been found in patients with Addison's disease who have been given excessive amounts of desoxycorticosterone acetate, in Cushing's syndrome, and in patients who are administered large amounts of adrenocorticotropic hormone, cortisone, or Artisone for any of the many reasons that these drugs are being administered today.

Repeated injections of adrenalin may lower the potassium concentration in an as yet unexplained manner, perhaps through the pituitary-adrenal mechanism, perhaps through a direct action on carbohydrate metabolism.

It has been postulated in unofficial reports, though not yet proved, that mercurial diuretics administered frequently over long periods of time may cause a state of hypopotassemia as well as a clinical picture of hyponatremia.

HYPERPOTASSEMIA

The clinical picture associated with marked increases in serum potassium does not become manifest until the serum potassium reaches extremely high levels. This consists of paralysis of striated muscle all over the body just as in hypopotassemia,[11] cardiac toxicity, electrocardiographic changes, and cardiac standstill, with unknown alterations of cellular metabolism. However, electrocardiographic manifestations which are supposedly pathognomonic of this state become evident at lower levels of hyperpotassemia[28, 31] (fig. 1). It has been found that no cardiotoxic changes occur below 6.8 milliequivalents per liter, whereas cardiotoxic changes are always found above 7.8 milliequivalents per liter; specific cardiographic changes can be expected at the various levels of hyperpotassemia. Between 6 and 8 milliequivalents of potassium per liter the T wave becomes tall, narrow, and peaked; between 8 and 10 milliequivalents the ST segment becomes depressed; at 10 milliequivalents an increase in the width of the QRS complex with intraventricular block occurs; between 9 and 11 milliequivalents there is a loss of P waves; and at 14 to 16 milliequivalents there is complete disintegration of the QRS complex with cardiac arrest in dias-

tole. Of importance is the fact that the QT interval is not prolonged as it is in hypopotassemia and hypocalcemia. The most important and the most constant observations in the sublethal range of hyperpotassemia are the T wave changes and the prolongation of the QRS complex.

Causes of Hyperpotassemia

Excessive Potassium Administration.—For many years, before the toxic effects of potassium were known, it was the custom to administer large amounts of potassium salts for various therapeutic purposes. It was not uncommon for a person with myasthenia gravis to receive anywhere from 10 to 40 Gm. of potassium chloride daily. This was given (with or without prostigmine) because of its anticholinesterase action at the myoneural junction and because it was reputed to give good results with no untoward effects. Potassium salts have also been used for their diuretic effect in doses of 10 Gm. of potassium chloride. They have also been employed in rather large dosage to treat Ménière's disease.

Six or seven years ago, before the advent of penicillin, it was the vogue among some physicians to use potassium carbonate as an alkalinizing agent together with sulfonamides. This was of particular value whenever the infection to be treated was complicated by congestive heart failure, and the sodium ion was therefore feared. Patients received 2 Gm. of potassium carbonate every four hours with supposedly no untoward effects.[26]

How much undetected toxicity resulted from these administrations can only be surmised. Certainly some of these patients, particularly those with renal disease, must have developed hyperpotassemic syndromes. That the administration of 5 Gm. of potassium salt orally will cause hyperpotassemia with cardiographic changes has been definitely proved in an experiment on normal persons.[19] These electrocardiographic alterations together with simultaneous serum potassium elevation were detected ninety minutes after the oral ingestion of potassium in several volunteer subjects. Others state that toxic effects will result only from the administration of much larger amounts or in the presence of renal damage.

Hemolysis.—Investigative work has been done with the injection of saponin into rabbits and the sudden causation of intravascular hemolysis of large amounts of blood.[33] Liberation of red blood cells potassium occurred in sufficient amount to cause elevation of the serum potassium, with electrocardiographic changes, cardiac dilatation, and death.

This experiment arouses speculation as to the possible cause of death in erythroblastosis fetalis, transfusion reactions, and other acute hemolytic diseases occurring in human beings. Since 100 cc. of red blood cells contain approximately 420 mg. of potassium, hemolysis of 1 pint of blood will cause the sudden liberation into the blood stream of about 1 Gm. of potassium. Obviously, impaired renal function, if already present, will augment the hyperpotassemia caused by an acute hemolytic process and increase the possibilities of cardiotoxicity.

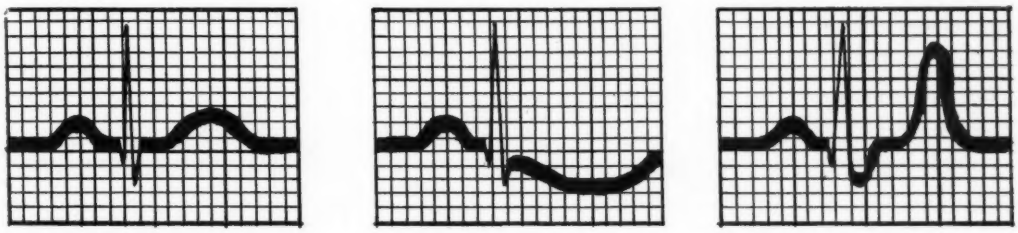

FIG. 1. Drawings showing the typical electrocardiographic tracing in normal persons and in hypopotassemia and hyperpotassemia. Note the inverted T wave, the depressed ST segment, and the prolonged QT interval caused by the wide T wave in hypopotassemia, and the narrow, tall T wave and wide QRS complex in hyperpotassemia.

Burns, Shock, Crush Syndrome.[12]—Surgical conditions which cause shock, such as severe burns and crush wounds, will notably also cause an elevation of serum potassium. This is because the damaged cells lose the selective permeabilities of their cell membranes and allow large amounts of sodium and water to enter the cells and large amounts of potassium to leave the cells and enter the extracellular fluids. In addition, uninjured cells in other parts of the body also take up sodium and water and release potassium; this results from the histamine-like reaction of toxic substances elaborated from the site of tissue damage. Thus, the serum potassium has been observed to rise to twice normal levels in instances of shock.

Addison's Disease.—In Addison's disease, as a result of a deficiency of cortical steroids, particularly those elaborated by the normal zona glomerulosa, there is an elevation of serum potassium. These steroids, if in excess, will cause elevation of the serum sodium

level with hypopotassemia; conversely, if these hormones are not present, the serum sodium level will fall, and the potassium level rise. Inadequate renal excretion of potassium, contraction of the volume of extracellular fluid, and inability of cells to take up potassium contribute to the hyperpotassemia seen in adrenal cortical deficiency. Experimentally, it has been noted that adrenalectomy in dogs can result in lethal potassium levels.

THERAPY

Potassium, when necessary, is most safely and best given orally, provided the patient's condition permits oral administration. Oral therapy is given with potassium chloride or acetate in doses of 1 to 2 Gm. hourly in tablet form or dissolved with fruit juices; or the potassium may be taken in potent beef broth or meat extracts, such as Valentine's meat extract, which contains 1,400 milliequivalents of potassium per liter.

If parenteral administration is necessary, potassium can be given subcutaneously using Darrow's solution (.26 Gm. of potassium chloride per 100 ml. of a sodium lactate-sodium chloride solution) or isotonic potassium chloride solution (1.14 Gm. per 100 ml.). These are given in 500 ml. amounts at a rate of 60 to 120 drops per minute, but always under electrocardiographic and laboratory control. Great caution must be used in all patients receiving intravenous potassium, but particularly in those with impaired renal function, oliguria, or anuria.

Hyperpotassemia may be treated by the administration of saline solution, intravenous glucose, and insulin, which mobilizes potassium in the liver, and a low potassium diet.

STUDIES WITH SPECTROPHOTOMETER

Studies of sodium and potassium levels in human blood serum as measured by the Beckman spectrophotometer with flame attachment reveal useful data (table 1).

The setting up of the apparatus and its attachments are somewhat complicated and are best accomplished with the help of the manufacturer or someone else familiar with its operation. The manipulation and adjustment of the apparatus is fully described in the manufacturer's bulletin.

The technique of analysis employed in this laboratory is the unpublished method of Albert Sample, Ph. D., of the Bryn Mawr Hospital, Philadelphia. Aqueous stock standards of calcium, magnesium, sodium, and potassium contain all of these elements in approximately the concentration of normal serum. Others recommend the preparation of stock standards which contain in addition gelatin to balance the viscosity of the serum, phosphates, sulfates, glucose, and cholesterol because of the effect these substances have upon the transmittance values of sodium and potassium. With the high dilutions of the stock standards and the serum recommended by Sample the depression is apparently not great for either sodium or potassium transmittance. The depression of transmittance values for calcium is much greater and must

TABLE 1.—*Preliminary Studies on Sodium and Potassium Levels in Human Blood Serum Measured by the Beckman Spectrophotometer with Flame Attachment.*

Case	Date	Diagnosis	Blood Sugar (mg. %)	Plasma CO_2 (vol. %)	Serum K (mEq/L)	Serum Na (mEq/L)	Medication
			NORMAL VALUES				
1					4.07	136.1	
2					5.08	133.4	
3					4.23	140.6	
4					4.67	127.7	
5					4.18	126.8	
6					4.30	127.7	
7					3.89	137.7	
8						144.9	
9						147.8	
			ABNORMAL VALUES				
10	3-15	Diabetes	251	32	3.21	162.2	KCl after coma
11	3-22 (4 p. m.)	Diabetic coma	720	10	2.77	143.5	KCl after coma
	3-22 (9 p. m.)				2.72	157.9	
	3-23 (9 a. m.)				3.26	157.9	
12	2-15	Coronary occlusion			5.78	123.4	
13	3-3	Uremia			6.32	120.9	
14	2-24	Gastric fistula			3.54		Darrow's solution
	3-4				2.87		
	3-7				2.79		
	3-11				3.35		
	3-22				3.01		
	3-29				3.26		

be compensated for by the addition of some or all of the substances mentioned. Dilution of serum for potassium determination is 1:100 and for sodium 1:1,000. As may be seen in table 1 values found for normals are within or slightly below the accepted normal range.

The intensity of the spectral lines depends first upon the concentration of the element heated in the flame. This intensity will be altered by the heat of the flame governed by the ratio of gas and oxygen pressure, the amount of atomized material determined by the air pressure exerted on the atomizer, the bore of the capillary of the atomizer, and the viscosity and temperature of the solution. These points are all well considered in the technical bulletin and by Mosher and his colleagues.[23] The one great source of error is the tendency of the fine capillary to become clogged with extremely small particles suspended in the solution; the settings of the apparatus once made rarely require readjustment. Failure to obtain check readings is usually due to clogging. Removal of the atomizer and reversing the flow of air through it quickly dislodges the offending particles. The laboratory technician may be trained to utilize this spectrophotometer successfully.

The settings of gas, oxygen and air pressure, and wave length must be determined for each instrument and for each element under examination. The settings generally recommended should be made and then with a working standard solution being sprayed into the flame, notation is made of the effect on the meter needle produced by lowering or raising the pressures slightly. Movement to the left indicates increased transmittance while a movement to the right indicates diminished transmittance. Maximum transmittance is desired. Our settings vary little from those of Sample except for the slit setting for potassium, which is lower, probably because of a more sensitive phototube.

SUMMARY

The basic physiology of sodium and potassium is reviewed, together with the clinical and electrocardiographic changes which occur with hypopotassemia and hyperpotassemia. Causes for the latter two conditions and methods of therapy are discussed. Preliminary studies on serum sodium and potassium measured with the Beckman spectrophotometer with flame attachment are reported.

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TETANUS SEEN IN BRACKENRIDGE HOSPITAL OVER A FIVE YEAR PERIOD

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NO doubt tetanus is a much more common disease in the South and Southwest than is generally acknowledged. Even with the present preventive and improved, though inadequate, therapeutic measures the mortality remains high.

Graves[5] in 1930 reviewed a total of 813 cases of tetanus in New Orleans. Vinnard[14] reviewed 352 cases of tetanus treated in Charity Hospital of Louisiana in the ten year period between 1934 and 1944. Gessler[4] reviewed 121 cases of tetanus seen in four hospitals in Nashville, Tenn., between 1933 and 1944. These papers constitute three of the largest series reported in the United States and all showed a mortality rate of 30 per cent or more.

REVIEW OF CASES

During the period between September 1, 1944, and September 1, 1949, 18 cases of tetanus were treated at the Brackenridge Hospital in Austin, Texas. The total deaths numbered 9. However, by excluding the patients who died within the first thirty-six hours and could be considered as moribund upon admission, as Vinnard[14] did, the net deaths numbered only 5. No apparent residual effect was recorded for the 9 patients who recovered.

Patients seen during the five year period ranged in age from 5 days to 76 years; 72 per cent were 10 years old or less.

Two patients were white, 9 were Latin Americans, and 7 were Negroes. This racial distribution is in line with the large number of Latin American and Negro patients seen at Brackenridge, the city hospital.

The incubation period was known in 11 cases and ranged from three days to two months. Of these, 8 patients related onset of symptoms to an injury of the lower extremity. Two others related their onset to a nail injury of the wrist and 1 to an infected cord stump. There was no correlation between the duration of the incubation period and the fatality of the disease in these cases.

Patients who died were hospitalized from one to fourteen days, whereas the length of hospitalization for those recovering ranged from eight days to 145 days.

Clinical Laboratory Signs.—Clinical observations made most frequently on admission of the patients were stiffness of the jaw and dysphagia. Two patients were admitted because of convulsions and 2 with the chief complaint of abdominal pain and cramping. Only 4 of the patients had a leukocyte count of less than 10,000 per cubic millimeter of blood on admission and 1 had a count of 40,000. Twelve patients had counts ranging from 10,000 to 15,000 leukocytes per cubic millimeter of blood on admission with normal differentials. Cultures were taken on 9 patients, and they were reported positive for Clostridium tetani in 2 cases. The 2 patients with positive cultures recovered, whereas 6 of the patients with reported negative cultures died.

Complications.—All of the patients who died had some stage of bronchopneumonia or pulmonary edema. Tracheotomy was done on only 2 patients, both of whom continued a downhill course and died. One patient seen in 1946 developed laryngospasm and died before a tracheotomy could be performed. Other complications included 1 severe case of herpes simplex, 1 case of pyelitis, and 1 of acute myocardial toxicity with congestive failure.

Treatment.—Tetanus antitoxin was administered in large doses to all patients. Both the intravenous and intramuscular routes were used in this series; none of the patients received intrathecal[9] or intrasternal[13] antitoxin. No record was available as to whether or not prophylactic tetanus antitoxin or a toxoid booster was given at the time of injury. All patients with a relatively short incubation period and an evident lesion received radical care of the wound to prevent further toxin formation. Only 3 of the patients had the tissue surrounding the wound infiltrated with antitoxin; 2 of these died from pulmonary complications and the third recovered.

Thirteen of the patients received large doses of penicillin;[1, 3, 11] 3 received combined penicillin and sulfadiazine therapy. Since these cases were seen aureomycin has been used to combat secondary pulmonary infections because of its wider antibacterial spectrum.[12]

All the patients were kept in cool, dark, quiet rooms and sedation maintained through one or a combination of drugs. Avertin was used in 3 cases, paraldehyde in 5 cases, chloral in 2 cases, and the barbiturates in some form in all cases. One-third of the patients seen after May 1, 1948, received large doses of vitamin K in an attempt to minimize pulmonary bleeding; however, the number of cases followed was too small to draw any definite conclusions. Two patients received curare[6]; 1 survived and the other died.

In all cases the treatment covered methods of combating convulsions, of combating toxin formation, of neutralizing toxin already formed and disseminated, and of preventing and combating common complica-

tions. These four basic considerations in treatment have been reviewed in detail by Costello.[2] The details of treatment varied in each case, and evaluation of the treatment received by those surviving and those dying is impossible. The outcome seemed to depend to a large measure on the severity of the disease and the ability to control symptoms and maintain life during the acute, fulminating period of the disease.

Distribution of Cases.—The number of cases varied greatly with each year. Only 1 patient received treatment each year in 1944, 1945, and 1949; 3 patients were treated in 1947; 4 in 1946; and 8 in 1948.

All but 1 of the patients treated came from within the city limits of Austin. A majority of the patients lived near the railroad tracks which pass through Austin. Seven lived within one block of the tracks, 2 within two blocks, 3 within four blocks, 1 within five blocks, and 3 within six blocks. Twelve of the patients came from the east side of town, and 2 of these cases occurred within an area of approximately a square mile. The factor of soil contamination of wounds always must be considered, and when such a distribution of cases as is reflected here occurs within a city, the source of soil contamination within this area must be questioned.

Prevention.—The prevention of tetanus rests on two major principles: (1) adequate care of all wounds and (2) establishment of either active or passive immunity as the case demands. The present practice of giving antitoxin to all wounded persons regardless of previous inoculation with toxoid has been challenged by some[4] and vindicated by others.[8] In this small series it is apparent from the histories that many of the patients did not obtain immediate medical care for their injury and were treated for the first time after the characteristic signs and symptoms of tetanus had developed.

SUMMARY AND CONCLUSIONS

This report adds 18 cases of tetanus to the literature. Specific measures and antibiotics appeared to be of little importance in the treatment unless used in association with intensified supportive measures, and even then the mortality rate remained high. Ten of the patients reported came from a relatively small section of Austin. Further investigation of the soil from this area plus an intensified program of active immunization for residents of this area seems indicated.

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P. O. Box 429.

Cancer Program Material Offered

The American Cancer Society, Texas Division is cooperating with county medical societies throughout the state in putting on demonstration tumor clinics. Upon request, the cancer society will supply a team of physicians to present a patient suffering from cancer and to discuss the case from the viewpoints of the surgeon, pathologist, radiologist, internist, and other specialists who might contribute to the treatment.

Almost 1,000 physicians have been present at such teaching conferences in recent weeks, and in several instances the demonstrations have given impetus to the establishment by county medical societies of tumor clinics to be held regularly in areas which previously have not had them.

In addition to supplying programs of the clinic demonstration type, the American Cancer Society through its Texas Division office makes available motion pictures, lantern slides, and other educational material appropriate for use in medical meetings.

Laboratory Training in Communicable Diseases

The United States Public Health Service has released a schedule of laboratory training courses to be given by the Communicable Disease Center, Chamblee, Ga., from January 1 to December 31, 1951. General topics to be covered are as follows: bacterial, parasitic, enteric, mycotic, venereal, and virus diseases and tuberculosis and rabies. Information and application forms should be requested from the Officer in Charge, Laboratory Training Services, Communicable Disease Center, P. O. Box 185, Chamblee, Ga.

Baylor Hospital Wins Courtesy Award

A campaign of courtesy at Baylor Hospital, Dallas, has won first award in a contest sponsored by the American Hospital Association, reports the Dallas *News*. The contest was planned to reward hospitals for a "program, technique, or routine" of administration which has proved successful. Of 106 entries from over the nation, Baylor's war on discourtesy was judged the best, winning for the institution a $100 government savings bond.

CASE REPORTS

CHOLESTEROL METABOLISM AND DEGENERATIVE DISEASES OF THE EYE

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THE examination of several patients seen in consultation because of eye involvement revealed that the blood cholesterol level was elevated. In 1948 a woman who had a retinal detachment of idiopathic origin was seen; the fundus of the good eye was in such a state of lipoid degeneration, or circinate retinopathy, that I feared for the good eye. A complete clinical investigation was made, and the significant findings were an elevated blood cholesterol level of 283 mg. per 100 cc. of plasma and a lowered metabolism of — 18 per cent. Wondering if this could have an influence on the course of her retinopathy, I reviewed the literature on the subject since 1946.

Of cholesterol it may be said that little is known except that it is concerned with absorption and metabolism of fat.[8] It is a sterol, and sterols are hydroaromatic alcohols, have no close chemical relation to fats, and are unsaponifiable. They are soluble in the same manner as fats, and in America it is acceptable to class the sterols with the lipids. Cholesterol and cholesterol esters in chemical structure appear to be related to bile salts, vitamin D, certain male and female sex hormones, and certain hormones of the suprarenal cortex. Physiologically there is no direct evidence of such close relationship.

Cholesterol has a wide range of normal in blood plasma.[9] The usually accepted range is from 140 to 250 mg. per 100 cc. There are increased values in chronic and acute nephritis, and it is of diagnostic importance in chronic nephrosis, diabetes, lipemia, pregnancy, cholelithiasis, and hypothyroidism. Duke-Elder[2] stated that a mild hypercholesterolemia has been reported by several observers to exist in some cataractous patients, whereas in others the blood cholesterol is normal. That general metabolic conditions have an influence in the determinations of cataract has always seemed obvious, both on account of the bilaterality of the condition and because of its tendency to develop in conditions of ill health. He further stated[3] that accumulations of lipid material may be found in the uveal tract in experimental lipoidosis, brought about by a cholesterin-rich diet.

It has long been recognized that there are cholesterin deposits in the coronary arteries, and this phase of the problem has been widely investigated. Burns[1] attempted to show that there is an individual tolerance to cholesterol intake. It is generally accepted that there is an elevation in cholesterol in coronary occlusion, and it has been noted in persons who present no other factors that would raise the cholesterol level. It is also known that the abnormal level can be lowered by diet. By diet the reduction is slow; however, in the presence of a low metabolism and with the addition of thyroid, the decrease is more rapid. Duke-Elder[4] suggested that circinate retinitis occurs on a cardiovascular or metabolic basis and is related to vascular sclerosis.

The work that is being done by Gallois[5] on the relationship of serum cholinesterase and glaucoma, the anticholingeric action of di-isopropyl fluorophosphate (DFP),[6] and a paper by Laborit and Morand[7] on the vascular syndromes and cholinesterase, all point to the importance of acquiring a better working knowledge of cholesterol and its esters.

CASE REPORTS

CASE 1.—Miss R. R., aged 66, was first seen February 21, 1950, her chief complaint being that her glasses, fitted in October, 1949, were not satisfactory. Her vision in the right eye was 20/70, corrected to 20/60 and Jaeger's test type 9, blurred. Vision in her left eye was 20/50, not corrected with glasses, and Jaeger's test type 9, blurred. Tension in each eye was 18 mm. of mercury. A fundus examination revealed lens opacities scattered throughout the cortex. Throughout each retina there was much degeneration of a large, coalescent drusen character, associated with grade 1 sclerosis of the arterioles. Her metabolic rate at that time was — 19 per cent and the blood cholesterol level was 398 mg. per 100 cc. of plasma. She was placed on thyroid medication and a low-cholesterol diet.

The patient was seen again April 3, at which time her vision with the same glasses was 20/50 in the right eye and 20/30 in the left. The Jaeger reading was type 6 for the right eye and type 3 for the left, and the patient stated that the test type was clear. There were no appreciable changes in the retinal lesions, none of which involved the maculas. Her metabolic rate had not changed and the dosage of thyroid was increased. The blood cholesterol level, however, had come down to 240 mg. per 100 cc. of plasma.

CASE 2.—T. E. J., a 59 year old white man, was seen June 14, 1949. His vision without glasses was 20/200 in the right eye and 20/60 in the left. His corrected vision was 20/25 in each eye. The fundus examination revealed moderate increase in reflex and about a grade 1 to 2 sclerosis of the arterioles. There were many drusens in each macular area.

From the Department of Ophthalmology, Scott and White Hospital. Read before the Section on Eye, Ear, Nose, and Throat, State Medical Association of Texas, Annual Session, Fort Worth, May 3, 1950.

The metabolic rate was 0 per cent and the blood cholesterol level 226 mg. per 100 cc. of plasma. The patient was placed on a low-cholesterol diet. When he was seen again September 16, his vision without glasses was 20/80 in the right eye and 20/40 in the left. With glasses his vision was 20/20 in each eye. There was no appreciable change in the fundi. His blood cholesterol level had dropped to 188 mg.

CASE 3.—Mrs. J. V. G., a 42 year old white woman, was seen July 15, 1949. She stated that her vision had started blurring in the right eye for both distance and near in 1939, and that she had not had a physical examination for some time. Vision in the right eye without glasses was 20/70 and in the left eye 20/20. A slit lamp examination revealed considerable opacities in the cortex of each lens with a number of globules. She had a metabolic rate of — 23 per cent and a blood cholesterol level of 249 mg. per 100 cc. of plasma. A tentative diagnosis of metabolic cataracts was made.

The patient was examined again March 17, 1950, at which time she stated that her vision was much clearer and that she had not been using her glasses, although her manifest vision was the same. There had been no appreciable change in the cataracts and no increase in loss of vision. The metabolic rate was — 14 per cent and the blood cholesterol level 214 mg. per 100 cc. of plasma.

CASE 4.—Mrs. L. S., a 56 year old woman, was seen January 26, 1950, with the minor complaint of her eyes burning and feeling irritated. Her corrected vision was 20/30 in the right eye and 20/25 in the left. She could read Jaeger's test type 9 with each eye corrected. A fundus examination showed scarring of the maculas and crystalline deposits in the retinas, not quite so marked in the left eye as in the right. There was a grade 3 sclerosis of the retinal vessels. Her metabolic rate was +16 and the blood cholesterol level was 346 mg. per 100 cc. of plasma. This was her only visit and she has not been reexamined.

SUMMARY

Attention is called to the possible relationship of cholesterol metabolism and degenerative diseases of the eye, primarily of the retina. The literature and several case histories relative to this subject are reviewed.

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ABSTRACT OF DISCUSSION

DR. R. M. JOHNSON, Houston: I saw a patient last week from another part of the state who comes under this discussion. She had been told that her condition was metabolic and nothing could be done. It is going to be interesting to see if thyroid and diet will improve her condition.

The essayist has covered the literature from 1946, which includes the essentials, but a few other points in review might be helpful. Wolff reported that cholesterol crystals which are rhomboid become needle-like on recrystallizing after being dissolved in ether. This explains why needle forms are found and why oval spaces are left after the tissue is treated with alcohol-ether solution. The role of the reticulo-endothelial system has been emphasized by Keiner in Niemann-Pick's, Gaucher's, and Schüller-Christian-Hand's disease in producing cholesterol deposits. Almeida concluded that retinal spots in the macular region, believed to be fatty, are caused by increased cholesterol content in the blood, but Joseph Igersheimer thought this doubtful. According to others, cholesterol deposits are found in the eyes and lids and as senile arcs in the cornea, and cholesterol is thought to cause widely varying ocular troubles associated with headaches. This hypothesis is based on a high serum cholesterol content and symptomatic improvement on proper diet.

As stated by Dr. McKay, experimental lipoidosis has been brought about by cholesterin-rich diet. Dr. Ray K. Daily, reviewing the literature from 1944 to 1946, stated that 63 per cent of guinea pigs kept on a scorbutigenic diet for from eighteen to twenty-one days developed cholesterosis lentis.

Chest Disease Essay Contest

A cash prize of $250 will be given for the best original contribution, preferably by a young investigator, on any phase relating to chest disease by the Board of Regents of the American College of Chest Physicians. The award and a certificate of merit will be given at the annual meeting of the College in Atlantic City, June 7-10, 1951.

The competition is open to contestants of other countries as well as those residing in the United States. The College reserves the right to invite the winner to present his essay at the annual meeting and to publish it in the College's official publication, *Diseases of the Chest*.

Five copies of the manuscript should be submitted not later than April 1, 1951, to the Executive Office of the College, 500 North Dearborn Street, Chicago 10. Additional details may be obtained from that source.

Baylor Hospital Given Bequest

An estate of approximately $1,000,000 has been divided equally between Baylor Hospital and Buckner Orphan's Home, two Dallas Baptist institutions, according to the Brownwood *Bulletin*. The estate of the late W. H. Thomas, a banker, went to the two institutions upon the death this year of his daughter, Mrs. May Thomas Taylor.

Houston Course in Atomic Medicine

In the first such regional conference 754 persons with high priority status met recently with Atomic Energy Commission experts at the Veterans Administration Hospital, Houston, for a series of ten lectures on means of coping with casualties caused by atomic warfare. Sponsoring organizations were Baylor Medical College, Veterans Hospital, and the Organized Reserve Corps, states the Houston *Chronicle*.

NEWS

COMING MEETINGS AND CLINICS

State Medical Association of Texas, Galveston, May 1-2, 1951. Dr. William M. Gambrell, Austin, Pres.; Tod Bates, 700 Guadalupe St., Austin, Executive Secy.

American Medical Association, Atlantic City, June 11-15, 1951. Dr. Elmer L. Henderson, Louisville, Ky., Pres.; Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secy.

NATIONAL AND REGIONAL

American Academy of Allergy, New York, Feb. 5-7, 1951. Dr. Theodore L. Squier, Milwaukee, Pres.; Mr. James O. Kelley, 208 E. Wisconsin Ave., Milwaukee 2, Executive Secy.

American Academy of Dermatology and Syphilology. Dr. Earl D. Osborne, Buffalo, N. Y., Pres.; Dr. John E. Rauschkolb, 25 Prospect Ave., N. W., Cleveland 15, Secy.

American Academy of General Practice, San Francisco, March 19-22, 1951. Stanley R. Truman, Oakland, Calif., Pres.; Mr. Mac F. Cahal, 406 W. 34th St., Kansas City 2, Executive Sec'y.

American Academy of Neurological Surgery. Dr. William S. Keith, Toronto, Canada, Pres.; Dr. T. C. Erickson, 1300 University Ave., Madison 5, Wis., Secy.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 14-18, 1951. Dr. J. MacKenzie Brown, Los Angeles, Pres.; Dr. W. L. Benedict, 100 First Ave. Bldg., Rochester, Minn., Secy.

American Academy of Pediatrics, Toronto, Oct. 20-25, 1951. Dr. Paul W. Beaven, Rochester, N. Y., Pres.; Dr. C. G. Grulee, 636 Church St., Evanston, Ill., Secy.

American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951. Dr. Alfred Blalock, Baltimore, Pres.; Dr. Brian Blades, 901 23rd St. N. W., Washington, D. C., Secy.

American Association of Genito-Urinary Surgeons, Skytop, Pa., May 16-18, 1951. Dr. Roger C. Graves, Boston, Pres.; Dr. Norris J. Heckel, 122 S. Michigan Ave., Chicago 3, Secy.

American Association of Obstetricians, Gynecologists, and Abdominal Surgeons, Hot Springs, Va., Sept. 6-8, 1951. Dr. James K. Quigley, Rochester, N. Y., Pres.; Dr. William F. Mengert, 2211 Oak Lawn Ave., Dallas, Secy.

American Cancer Society. Dr. Guy Aud, Louisville, Ky., Pres.; Mr. M. R. Runyon, 47 Beaver St., New York, Exec. Vice-Pres.

American College of Allergists, Chicago, Feb. 11-14, 1951. Dr. John H. Mitchell, Columbus, Ohio, Pres; Dr. Fred W. Wittich, 423 La Salle Medical Building, Minneapolis 2, Secy.

American College of Chest Physicians, Atlantic City, N. J., June 7-10, 1951. Dr. Louis Mark, Columbus, Ohio, Pres.; Mr. Murray Kornfeld, 500 N. Dearborn St., Chicago 10, Executive Secy.

American College of Physicians, St. Louis, April 9-13, 1951. Dr. William S. Middleton, Madison, Wis., Pres.; Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Secy.

American College of Radiology, Atlantic City, June 10, 1951. Dr. C. Edgar Virden, Kansas City, Mo., Pres.; Mr. W. C. Stronach, 20 N. Wacker Drive, Chicago 6, Executive Secy.

American College of Surgeons, San Francisco, 1951. Dr. Frederick A. Coller, Ann Arbor, Mich., Pres.; Dr. Paul B. Magnuson, 40 E. Erie St., Chicago 11, Secy.

American Congress of Physical Medicine, Denver, Sept 3-8, 1951. Dr. Arthur L. Watkins, Boston, Pres.; Dr. Richard Kovacs, 2 E. 88th St., New York 28, Secy.

American Dermatological Association, Hot Springs, Va., May 23-26, 1951. Dr. Henry E. Michelson, Minneapolis, Pres.; Dr. L. A. Brunsting, 102 2nd Ave., S. W., Rochester, Minn, Secy.

American Gastro-Enterological Association, Atlantic City, June 8-9, 1951. Dr. John G. Mateer, Detroit, Pres.; Dr. Dwight L. Wilbur, 655 Sutter St., San Francisco, Secy.

American Gynecological Society, New York, May 7-9, 1951. Dr. Frederick Irving, Brookline, Pres.; Dr. Norman F. Miller, 1313 E. Ann St., Ann Arbor, Mich., Secy.

American Hospital Association. Dr. Charles F. Wilinsky, Boston, Pres.; Mr. George P. Bugbee, 18 E. Division St., Chicago, Executive Secy.

American Laryngological, Rhinological, and Otological Society, White Sulphur Springs, W. Va., May 6-8, 1951. Dr. Louis H. Clerf, Philadelphia, Pres.; Dr. C. S. Nash, 277 Alexander St., Rochester 8, N. Y., Secy.

American Neurological Association. Dr. Henry W. Woltman, Rochester, Minn., Pres.; Dr. H. Houston Merritt, 710 W. 168th St., New York 32, Secy.

American Ophthalmological Society, White Sulphur Springs, W. Va., June 7-9, 1951. Dr. John H. Dunnington, New York, Pres.; Dr. M. C. Wheeler, 30 W. 59th St., New York 19, Secy.

American Orthopedic Association, White Sulphur Springs, W. Va., June 20-23, 1951. Dr. James S. Speed, Memphis, Pres.; Dr. C. Leslie Mitchell, Henry Ford Hospital, Detroit 2, Secy.

American Pediatric Society, Atlantic City, May 2-5, 1951. Dr. Bronson Crothers, Boston, Pres.; Dr. Henry G. Poncher, 1819 W. Polk St., Chicago 12, Secy.

American Proctologic Society, Atlantic City, June 7-10, 1951. Dr. Hoyt R. Allen, Little Rock, Pres.; Dr. W. Wendell Green, 1838 Parkwood Ave., Toledo 2, Secy.

American Psychiatric Association. Dr. John C. Whitehorn, Baltimore, Pres.; Dr. R. Finley Gayle, 501 E. Franklin St., Richmond, Va., Secy.

American Public Health Association. Dr. Lowell J. Reed, Baltimore, Pres.; Dr. R. M. Atwater, 1790 Broadway, New York 19, Secy.

American Society of Anesthesiologists, Washington, D. C., Nov. 5-8, 1951. Dr. Urban H. Eversoll, Boston, Pres.; Dr. J. E. Remlinger, Jr., 188 W. Randolph St., Chicago, Secy.

American Society of Clinical Pathologists, Chicago, Oct. 13-20, 1951. Dr. James B. McNaught, Denver, Pres.; Dr. Clyde G. Culbertson, Indiana University School of Medicine, Indianapolis, Secy.

American Surgical Association, Washington, D. C., April 11-13, 1951. Dr. Samuel C. Harvey, New Haven, Conn., Pres.; Dr. Nathan Womack, University of Iowa, Iowa City, Secy.

American Urological Association, Chicago, May 21-24, 1951. Dr. Thomas D. Moore, Memphis, Tenn., Pres.; Dr. C. H. deT. Shivers, 121 S. Illinois Ave., Atlantic City, N. J., Secy.

Association of American Physicians and Surgeons, Indianapolis, Oct. 4-6, 1951. Dr. Lawerence Shinabery, Fort Wayne, Ind., Pres.; Mr. Harry E. Northam, 360 N. Michigan Ave., Chicago 1, Executive Secy.

International College of Surgeons, U. S. Chapter. Dr. Custis Lee Hall, Washington, D. C., Pres.; Dr. Arnold S. Jackson, 1516 Lake Shore Drive, Chicago, Secy.

National Tuberculosis Association, Cincinnati, May 14-18, 1951. Dr. David T. Smith, Durham, N. C., Pres.; Dr. H. Stuart Willis, 1790 Broadway, New York 19, Secy.

Radiological Society of North America. Dr. Warren W. Furey, Chicago, Pres.; Dr. D. S. Childs, Medical Arts Bldg., Syracuse 2, N. Y., Secy.

Southern Medical Association, Dallas, Nov. 5-8, 1951. Dr. Curtice Rosser, Dallas, Pres.; Mr. C. P. Loranz, 1020 Empire Bldg., Birmingham, Ala., Secy.

Southern Psychiatric Association. Dr. R. Finley Gayle, Jr., Richmond, Va., Pres.; Dr. Newdigate M. Owensby, Medical Arts Bldg., Atlanta, Ga., Secy.

Southern Surgical Association. Dr. G. V. Brindley, Temple, Pres.; Dr. John C. Burch, 2112 West End Ave., Nashville, Tenn., Secy.

Southwest Allergy Forum, San Antonio, April 8-10, 1951. Dr. J. H. W. Rouse, San Antonio, Pres.; Dr. Boen Swinny, 224 Medical Arts Bldg., San Antonio 5, Secy.

Southwest Regional Cancer Conference. Secy., 209 Medical Arts Bldg., Fort Worth.

Southwestern Medical Association, El Paso, Fall, 1951. Dr. L. W. Breck, El Paso, Pres.; Dr. Wickliffe R. Curtis, First National Bank Bldg., El Paso, Secy.

Southwestern Surgical Congress, St. Louis, Sept. 24-26, 1951. Dr. Leo J. Starry, Oklahoma City, Pres.; Dr. C. R. Rountree, 1227 Classen, Oklahoma City 3, Secy.

Tri-State Medical Assembly. Dr. W. S. Terry, Jefferson, Pres.; Dr. James Harris, Marshall, Secy.

United States-Mexico Border Public Health Association, Los Angeles, April 4-6, 1951. Dr. Luis Arriaga Velez, Chihuahua, Mexico, Pres.; Dr. M. F. Haralson, 314 U. S. Court House, El Paso, Secy.

STATE

Texas Academy of General Practice, Houston, September 10-11, 1951. Dr. Andrew S. Tomb, Victoria, Pres.; Dr. B. H. Bayer, 104 E. Twentieth St., Houston, Secy.

Texas Academy of Internal Medicine. Dr. Samuel A. Shelburne, Dallas, Pres.; Dr. John S. Chapman, 3810 Swiss Ave., Dallas, Secy. Meetings restricted to members.

Texas Air-Medics Association, Galveston, April 30, 1951. Dr. D. P. Laugenour, Dallas, Pres.; Dr. C. F. Miller, 906 Medical Arts Bldg., Waco, Secy.

Texas Association of Obstetricians and Gynecologists, Galveston, Feb. 9-10, 1951. Dr. Howard Smith, Marlin, Pres.; Dr. George F. Adam, 4115 Fannin, Houston, Secy.

Texas Chapter, American College of Chest Physicians, Galveston, April 30, 1951. Dr. David McCullough, Kerrville, Pres.; Dr. Henry R. Hoskins, 514 Medical Arts Bldg., San Antonio, Secy.

Texas Club of Internists. Dr. Ghent Graves, Houston, Pres.; Dr. Hatch W. Cummings, Jr., Houston, Secy.

Texas Dermatological Society, Galveston, April 30, 1951. Dr. J. G. Brau, Dallas, Pres.; Dr. W. Harris Connor, 601 Medical Arts Bldg., Houston, Secy.

Texas Diabetes Association, Galveston, April 29, 1951. Dr. B. F. Smith, Houston, Pres.; Dr. W. N. Powell, W. Ave. F, Temple, Secy.

Texas Division, American Cancer Society, Fall, 1951. Mr. Frank C. Smith, Houston, Pres.; Mr. J. Louis Neff, 2307 Helena St., Houston 6, Executive Director.

Texas Heart Association, Galveston, April 30, 1951. Dr. George W. Parson, Texarkana, Pres.; Miss Roberta Miller, 411 Reserve Loan Life Bldg., Dallas, Executive Secy.

Texas Hospital Association, San Antonio, April 24-26, 1951. Mr. Roy Wilmesmeier, Houston, Pres.; Mrs. Ruth Barnhart, 2210 Main St., Dallas, Secy.

Texas Neuropsychiatric Association, Galveston, April 30, 1951. Dr. Martin L. Towler, Galveston, Pres.; Dr. James Blair, San Antonio, Secy.

Texas Orthopedic Association, Galveston, April 30, 1951. Dr. Louis Breck, El Paso, Pres.; Dr. Margaret Watkins, 3629 Fairmount St., Dallas, Secy.

Texas Pediatric Society, Houston, October, 1951. Dr. Thomas D. McCrummen, Austin, Pres.; Dr. M. C. Carlisle, 1410 Austin Ave., Waco, Secy.

Texas Public Health Association, Galveston, Feb. 18-21, 1951. Mr. Barnie A. Young, Austin, Pres.; Mr. Earle W. Sudderth, Dallas County Health Department, Court House, Dallas, Executive Secy.

Texas Radiological Society, Galveston, Jan. 19-20, 1951. Wayne D. Ramsey, Abilene, Pres.; Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth, Secy.

Texas Railway and Traumatic Surgical Association, Galveston, April 30, 1951. Dr. R. J. White, Fort Worth, Pres.; Dr. W. F. Parsons, First National Bank Bldg., Fort Worth, Secy.

Texas Rheumatism Association. Dr. Moise D. Levy, Jr., Houston, Pres.; Dr. Robert H. Mitchell, 210 Medical Arts Bldg., Fort Worth, Secy.

Texas Society for Mental Health, Galveston, March 8-9, 1951. Dr. Hamilton Ford, Galveston, Pres.; Mrs. Elizabeth F. Gardner, 1617 Watchhill Road, Austin 21, Executive Secy.

Texas Society of Anesthesiologists. Galveston, April 30, 1951. Dr. Russell Bonham, Houston, Pres.; Dr. Werner Hoeflich, 2301 Reba, Houston, Secy.

Texas Society of Gastroenterologists and Proctologists, Galveston, April 30, 1951. Dr. Carl G. Giesecke, San Antonio, Pres.; Dr. John S. Bagwell, Medical Arts Bldg., Dallas, Secy.

Texas Society of Ophthalmology and Otolaryngology. Dr. V. R. Hurst, Longview, Pres.; Dr. John L. Matthews, 929 Nix Professional Bldg., San Antonio, Secy.

Texas Society of Pathologists, Galveston, Jan. 28, 1951. Dr. Charles Phillips, Temple, Pres.; Dr. A. O. Severance, 205 Camden, San Antonio, Secy.

Texas Surgical Society, Galveston, April 2-3, 1951. Dr. Edward White, Dallas, Pres.; Dr. Truman G. Blocker, 927 Strand, Galveston, Secy.

Texas Tuberculosis Association, Dallas, April 13-14, 1951. Dr. Elliott Mendenhall, Dallas, Pres.; Miss Pansy Nichols, 208 E. Ninth, Austin, Executive Secy.

Texas Urological Society, San Antonio, Jan. 22, 1951. Dr. Hub E. Isaacks, Fort Worth, Pres.; Dr. John M. Pace, 428 Medical Arts Bldg., Dallas, Secy.

DISTRICT

Second District Society, Midland, March 13, 1951. Dr. James W. Rainer, Odessa, Pres.; Dr. Frank M. James, 1021 N. Whitaker Ave., Odessa, Secy.

Third District Society, Amarillo, April 10-11, 1951. Dr. Allen T. Stewart, Lubbock, Pres.; Dr. James T. Hall, 1302 Avenue Q, Lubbock, Secy.

Fourth District Society, Brady, November, 1951. Dr. S. Braswell Locker, Brownwood, Pres.; Dr. B. A. Hallum, Brady, Secy.

Fifth and Sixth Districts Society, Corpus Christi, July 6-7, 1951. Dr. E. King Gill, Corpus Christi, Pres.; Dr. Foy Moody, 1611 Fifth St., Corpus Christi, Secy.

Seventh District Society, Austin, Feb. 21, 1951. Dr. John F. Thomas, Austin, Pres.; Dr. George W. Tipton, 502 W. 15, Austin, Secy.

Eighth District Medical Society, March 23-24, 1951. Dr. Leonard Johnson, El Campo, Pres.; Dr. Robert Casey, Texas City, Secy.

Ninth District Society. Dr. L. E. Bush, Huntsville, Pres.; Dr. Lyman C. Blair, 1212 Rothwell, Houston, Secy.

Tenth District Medical Society, Port Arthur, March 3, 1951. Dr. A. J. Richardson, Sr., Jasper, Pres.; Dr. L. C. Heare, 221 Bluestein Bldg., Port Arthur, Secy.

Eleventh District Society, Henderson, Spring, 1951. Dr. Griff T. Ross, Mount Enterprise, Pres.; Dr. John M. Travis, Jr., Jacksonville, Secy.

Twelfth District Society, Temple, Jan. 9, 1951. Dr. W. K. Logsdon, Corsicana, Pres.; Dr. N. C. Smith, Hillsboro, Secy.

Thirteenth District Society. Dr. R. L. Daily, Wichita Falls, Pres.; Dr. S. W. Wilson, Medical Arts Bldg., Fort Worth, Secy.

Fourteenth District Society, Bonham, June, 1951. Dr. Mayo Tenery, Waxahachie, Pres.; Dr. L. W. Johnston, 502 W. College St., Terrell, Secy.

Fifteenth District Society, 1951. Dr. R. G. Granbery, Marshall, Pres.; Dr. H. O. Padgett, Marshall, Secy.

CLINICS

Dallas Southern Clinical Society, Dallas, March 26-29, 1951. Miss Betty Elmer, Medical Arts Bldg., Dallas 1, Executive Secy.

International Post-Graduate Medical Assembly of Southwest Texas, San Antonio, Jan. 23-25, 1951. Dr. John J. Hinchey, P. O. Box 2445, San Antonio, Secy.

New Orleans Graduate Medical Assembly, New Orleans, March 5-8, 1951. Dr. Woodard D. Beacham, Room 105, 1430 Tulane Ave., New Orleans 12, Secy.

North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls, Sept. 19, 1951. Dr. W. L. Powers, Hamilton Bldg., Wichita Falls, Chairman.

Oklahoma City Clinical Society Conference, Oklahoma City, Oct. 29-Nov. 1, 1951. Mrs. Muriel R. Waller, 512 Medical Arts Bldg., Oklahoma City 2, Executive Secy.

Post Graduate Medical Assembly of South Texas, Houston, July 23-25, 1951. Dr. Donald M. Paton, Secy., 229 Medical Arts Bldg., Houston.

UNIVERSITY OF TEXAS MEDICAL BRANCH

T. S. Painter, Ph. D., Austin, president of the University of Texas, gave the Sigma Psi Lecture on "Expanding Programs for Graduate Education" before the Galveston chapter November 20.

Chauncey D. Leake, Ph. D., vice-president of the University of Texas Medical Branch, spoke on "The History of Medical Ethics" at the installation dinner of Alpha Omega Alpha, honorary medical fraternity, at Southwestern Medical School of the University of Texas, Dallas, on November 10. President T. S. Painter of the University of Texas, Austin, presided at the dinner and was made an honorary member of the fraternity. Installing officers were Dr. Walter Bierring, Des Moines, president of the fraternity, and Dr. Josiah Moore, Chicago, secretary.

Dr. T. G. Blocker, Jr., professor of plastic and maxillofacial surgery at the Medical Branch, has arranged for demonstrations of the open air exposure method of treating extensive burns which have been attended by medical authorities from London, Edinburgh, and Stockholm.

Recent visitors at the Medical Branch were Dr. Karl Morgan, director of health physics, Oak Ridge, Tenn., who spoke on radioisotopes; Dr. H. W. Smith, Tulane University of Louisiana, New Orleans, who conducted a seminar on forensic problems in psychiatry; and Dr. J. N. Stannard, Department of Radiation-Biology, University of Rochester, Rochester, N. Y., who talked on the biological effects of radiation.

NEURO-OPHTHALMOLOGIST TO SPEAK

Dr. Alfred Kestenbaum, New York, internationally known neuro-ophthalmologist, will be the guest speaker of the Dallas Society for Ophthalmology and Otolaryngology at its meeting February 4-6 in Dallas. His subjects will be as fol-

lows: visual fields in lesions of the chiasmal region, physiology and pathology of the pupil, neuro-anatomy and topical diagnosis of the extraocular muscles, and paralysis of associated eye movement.

At one session cases will be presented for clinical discussion. Those who wish to present neuro-ophthalmological patients should notify Dr. Harold M. Block, Medical Arts Building, Dallas, in advance.

During the three-day meeting two luncheons and three dinners will be given. No registration fee will be charged. Physicians may write Dr. Block for reservations and additional information.

PERSONALS

Dr. Walter G. Rice, Dallas, immediate past-president of the Texas Association of Blood Banks, has been named to the staff of the St. Louis University School of Medicine, reports the San Antonio *Express.*

Dr. B. C. Bullen has been appointed chief medical officer of the Veterans Administration regional office in San Antonio, states the Johnson City *Record-Courier.* For the past two years he was chief of professional service at the Veterans Administration Hospital, Kerrville.

The Texas State Board of Health recently elected *Dr. George W. Cox* of Austin to his eighth consecutive two-year term as State Health Officer, the longest tenure in the more than seventy year history of the State Department of Health, according to the Alice *Daily Echo.* Members of the Board are *Drs. O. B. Kiel,* Wichita Falls, chairman; and *S. D. Coleman,* Navasota; *John H. Mitchell,* Tyler; *A. W. Rogers,* Corsicana; *T. C. Terrell,* Fort Worth; *B. E. Pickett,* Carrizo Springs; and *L. S. Oates,* Center.

Dr. R. Lee Clark, Jr., director and surgeon-in-chief of the M. D. Anderson Hospital for Cancer Research, Houston, delivered the tenth annual Barnard Lecture November 21 in St. Louis on "What Can We Do in Cancer Today?"

Dr. W. W. Schuessler, El Paso, attended the ninth National Assembly of Surgeons in Mexico City and spoke before the plastic surgery section November 22, reports the El Paso *Times.* He also attended the convention of the American Society of Plastic and Reconstructive Surgeons in Mexico City in November.

Dr. Paul R. Meyer, Port Arthur, has been elected president of the Port Arthur YMCA, comments the Port Arthur *News.*

Dr. John B. Cummins, Fort Worth, celebrated his ninety-second birthday on November 7, the Fort Worth *Press* comments. He has been a general practitioner for fifty-three years, forty-two of them in Fort Worth. This past January he was named General Practitioner of the Year by Tarrant County Medical Society.

Dr. A. J. Pope, La Feria, and his family were honored at a Kiwanis club banquet November 14 in La Feria, reports the Harlingen *Valley Morning-Star.* He was given a plaque commemorating his forty-four years' service as a practicing physician, twenty-one of which have been spent in La Feria.

Dr. D. J. Jenkins, Daingerfield, was honored by his home town when "Dr. Jenkins Day" was proclaimed November 9, states the Dallas *News.* A parade, concert, and program were held, and Dr. Jenkins was presented twenty-four yellow chrysanthemums for his sixty-three years of practice. Present at the ceremony were fourteen persons, including a set of twins, each representing a five-year span in his service. Dr. Joe Nichols, Atlanta, Fifteenth District councilor, spoke on "The Professional Life of Doctor Jenkins."

Dr. Lynn A. Bernard, Houston, and Miss Margaret Conrad were wed June 9, according to the *Bulletin* of the University of Texas Medical Branch Alumni Association.

Dr. Robert V. Murray, Austin, married Miss Claire Scott in Austin on September 26, reports the University of Texas Medical Branch Alumni Association *Bulletin.*

A girl was born recently to *Dr. and Mrs. E. Ivan Bruce, Jr.,* states the *Bulletin* of the University of Texas Medical Branch Alumni Association.

Recent parents of a boy are *Dr. L. U. Mewhinney* and *Mrs. Mewhinney (Dr. Ruth Allen),* Dallas, according to the *Bulletin* of the University of Texas Medical Branch Alumni Association.

Dr. and Mrs. James L. Johnson, Amarillo, recently became the parents of a girl, reports the Potter County Medical Society *Bulletin.*

Mrs. Lula Shelton, the widow of the late *Dr. E. P. Shelton* of Dripping Springs, who died in 1946, died in her Burnet home December 19, according to the Austin *American.*

Chicago Meetings of Interest

The eighteenth annual conference of the American College of Radiology will be held at the Palmer House, Chicago, Illinois, on February 10, 1951. The program, devoted to the radiologic aspects of civilian defense, will feature authorities on each phase of radiologic defense. Physicians and civilian defense leaders from each state who wish to attend may write for further information to Dr. John D. Camp, Chairman, Commission on Education, American College of Radiology, 20 North Wacker Drive, Chicago 6.

The National Conference on Medical Services will hold its twenty-fourth annual meeting the following day, February 11, at the Palmer House. The meeting will be of importance to administrative and elective personnel of state and county medical societies. Additional information may be obtained from Dr. R. E. Fitzgerald, Secretary, National Conference on Medical Service, 704 East Gorham Street, Madison 3.

The seventh annual meeting of the American College of Allergists will be held at the Edgewater Beach Hotel from February 12 to 14. Section meetings will be concerned with psychosomatic aspects of allergic diseases, pediatrics, allergies of the nose and throat, allergic diseases of the skin, and allergic aspects of rheumatism and arthritis. At a general session hay fever, asthma, and the newer drugs will be discussed.

For the first time this year the College is offering its post-collegiate instructional course on the three days preceding the annual conclave, from February 9 to 11. A fee of $35 will be charged. Dr. Fred Wittich, Secretary-Treasurer American College of Allergists, LaSalle Medical Building, Minneapolis, can supply further information.

Dermatologic Essay Contest

The American Dermatological Association is offering a prize of $300 for the best essay of original work not previously published relative to a fundamental aspect of dermatology or syphilology. Manuscripts typed in English with double spacing should be submitted in triplicate not later than February 1, 1951, to Dr. Louis A. Brunsting, Secretary, 102-110 Second Avenue, S. W., Rochester, Minn. Further information may be obtained from Dr. Brunsting.

Child Behavior Problems to Be Studied

Problems of child behavior will be considered in an institute February 15-17 at the University of Texas Medical Branch, Galveston, to which teachers, school physicians, and other interested persons are invited. Dr. Leo Kanner, director of the Children's Psychiatric Clinic at Johns Hopkins Hospital, Baltimore, will be guest speaker. Additional information may be obtained from Dr. Arild E. Hansen, Director, Child Health Program, University of Texas Medical Branch.

LIBRARY SECTION

PACKAGE SERVICE

The package library consists of collections of reprints and other periodical material on various subjects, prepared for lending to members of the Association. Request for packages should be addressed "Library, State Medical Association of Texas, 700 Guadalupe Street, Austin, Texas." Twenty-five cents in stamps should be enclosed with the request to cover postage and part of the expense of collecting the material. Packages are allowed to remain in the hands of the borrower for 14 days.

ACCESSIONS

The following additions were made to the Library during December:

Reprints received, 1,687.

Journals received, 266.

Books received, 19.

Preparation of Photographic Prints for Medical Publication, McComb; *Indications for and Results of Splenectomy,* Coller and others; *Problems in Cerebellar Physiology,* Moruzzi; *Visual Anatomy, Head and Neck,* Friedman; *Pharmacological Basis of Penicillin Therapy,* Beyer; *The External Secretion of the Pancreas,* Thomas; *Food Allergy,* Rinkel and Zeller; *Fainting, Physiological and Psychological Considerations, Engel,* Charles C. Thomas, Springfield, Ill.

The Other Side of the Bottle, Anderson, A. A. Wyn, Inc., New York.

Natural Childbirth, Goodrich, Prentice-Hall, Inc., New York.

Methods in Medicine, 2nd edition, revised, Dock and Herrmann; *Physiology of the Eye, Clinical Application,* Adler, C. V. Mosby Company, St. Louis.

Santa Claus, M. D., 1st edition, Bauer, Bobbs-Merrill Co., Inc., Indianapolis, New York.

Recent Advances in Nutrition, Cannon, University of Kansas Press, Lawrence, Kan.

Transactions of the American Therapeutic Society, Vol. XIVIII, 1947, Pottenger, editor, Commonwealth Press, Worcester, Mass.

The Rhesus Danger, Its Medical, Moral and Legal Aspects, 1st edition, McCurdy, William Heinemann, London.

Savill's System of Clinical Medicine, 13th edition, Warner, Williams and Wilkins, Baltimore.

Physicians' Desk Reference, Pharmaceuticals and Biologicals, Medical Economics, Inc., Rutherford, N. J.

Fifth Annual Report Life Insurance Medical Research Fund, George Grady Press, New York.

SUMMARY OF SERVICE

Local users, 41. Borrowers by mail, 117.
Items borrowed, 207. Packages mailed, 53.
Films loaned, 57.

New Texas Psychiatric Publication

The first issue of the new *Psychiatric Bulletin* has been distributed to 7,000 Texas physicians and a limited number of other physicians over the country. It is planned and edited principally by faculty and staff members of institutions in the Texas Medical Center, Houston. Similar in format to the *Cancer Bulletin* and illustrated in color, its editor is Dr. R. Lee Clark; its executive editor, Russell W. Cumley, Ph. D.; and its directing medical editor, Dr. Jack R. Ewalt. Art director is Joseph H. Schwarting.

MOTION PICTURE FILM LIBRARY

Motion picture films on medical subjects, 16 mm., both silent and sound, some in color, and suitable for either medical or lay audiences, are available for loan to county medical societies, hospital staffs, or individual physicians, on request. Borrowers will be required to pay only the cost of shipment of the films, by express, with insurance, and for any damage to films in the hands of the borrower.

Request for films should be addressed to "Motion Picture Film Library, State Medical Association of Texas, 700 Guadalupe Street, Austin, Texas." A list of available films, with descriptions, will be furnished on request.

The following motion picture films were loaned by the Film Library during the month of December:

Accent on Use (National Foundation for Infantile Paralysis)—Non Travis Hospital, Jacksonville.

Allergy (Mead Johnson)—Scott and White Memorial Hospitals School of Nursing, Temple.

A.M.A. Newsreel (American Medical Association, Department of Public Relations)—Gonzales County Medical Society, Gonzales.

Anemia, Erythroblastic (Mead Johnson)—Dr. R. W. Loveless, Bastrop, and Seton Hospital School of Nursing, Austin.

Anemias, The (Lederle Laboratories, Inc.)—Dr. R. W. Loveless, Bastrop.

Anesthesia, Regional (Winthrop Chemical Corp.)—Lamb-Bailey-Hockley-Cochran County Medical Society, Levelland.

Another to Conquer (Texas Tuberculosis Association)—Gonzales High School, Gonzales.

As Others See Us (American Hospital Association)—Wichita Falls Clinic Hospital, Wichita Falls.

Bleeding Tendency, Methods for Determination of (Mead Johnson)—Scott and White Memorial Hospitals School of Nursing, Temple.

Blood Transfusion (British Information Services)—College of Pharmacy, The University of Texas, Austin.

Breast Cancer, the Problem of Early Diagnosis (American Cancer Society)—Gray-Wheeler-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Medical Society, Pampa.

Breast Plastic: One-Stage Operation for Pendulous Breasts (Dr. Philip Thorek)—Drs. Hyslop and Hyslop, Del Rio, and Surgical-Medical Psychology Group, Houston.

Breast Self-Examination for Women (American Cancer Society)—Memorial Hospital, Sulphur Springs, and Surgical-Medical Psychology Group, Houston.

Bronchial Asthma (E. Fougera and Company)—Scott and White Memorial Hospitals School of Nursing, Temple.

Cardio-Vascular Anomalies, Congenital, Amenable to Surgery (Mead Johnson)—Karnes-Wilson Counties Medical Society, Karnes City.

Cataract Surgery (Dr. R. K. Daily)—Scott and White Memorial Hospitals School of Nursing, Temple.

Chest Diseases, Surgery in (British Information Services)—Nightingale Hospital Staff, Del Rio, and Parent-Teacher Association, San Felipe High School, Del Rio.

Cloud in the Sky (Texas Tuberculosis Association)—Elizabeth Nixson Junior High School, Amarillo.

Coming Home (Texas Tuberculosis Association)—Fisher County Tuberculosis Association, Roby, and Drs. Hyslop and Hyslop, Del Rio.

Dysmenorrhea, Primary (Searle and Company)—Wilson

Jones Hospital Staff, Sherman, and Medical and Surgical Clinic, Laredo.

Electric Shock Therapy in Depressions (Drs. Melbourne J. Cooper and James R. Blair)—Victoria-Calhoun-Goliad Counties Medical Society, Victoria.

Esophagogastrostomy, Supra-Aortic, for Carcinoma of the Midportion of the Esophagus (Dr. Philip Thorek)—Veterans Administration Hospital, Legion Branch, Kerrville.

Esophagus, Surgical Treatment for Carcinoma of the Lower End of (Dr. Philip Thorek)—Drs. Hyslop and Hyslop, Del Rio.

Extracellular Fluid, Introduction to (Mead Johnson)—Scott and White Memorial Hospitals School of Nursing, Temple.

Eyes for Tomorrow (Hurst Eye, Ear, and Throat Clinic)—Brackenridge Hospital School of Nursing, Austin.

Eyes, Your Children's (British Information Services)—Scott and White Memorial Hospitals School of Nursing, Temple.

Gastrectomy, Safer (Billy Burke Productions)—Medical and Surgical Clinic, Laredo.

Gastroscopy, Role of, in Gastric Pathology (Harrower Laboratory, Inc.)—Walker-Madison-Trinity Counties Medical Society.

Goodbye, Mr. Germ (Texas Tuberculosis Association)—General Science Students, Van Public Schools, Van.

Hemicolectomy for Carcinoma of the Right Side of the Colon (Dr. Philip Thorek)—Anderson-Houston-Leon Counties Medical Society, Palestine.

Hepatitis, Observation on (Mead Johnson)—Veterans Administration Hospital, Legion Branch, Kerrville.

Hidden Hunger (Swift and Company)—Adult Homemaking Classes, Gonzales High School, Gonzales.

Hypodermic Syringes and Needles: Their Care and Function (Becton, Dickinson and Company)—College of Pharmacy, The University of Texas, Austin.

Immunization Against Infectious Diseases (Lederle Laboratories)—Scott and White Memorial Hospitals School of Nursing, Temple.

Injuries, Athletic, Care and Prevention (Becton, Dickinson and Company)—Dr. B. L. Burditt, Del Rio, and Dr. V. M. Payne, Dallas.

Intracranial Injuries of the Newborn (Mead Johnson)—Walker-Madison-Trinity Counties Medical Society, Huntsville.

Let My People Live (Texas Tuberculosis Association)—Gonzales High School, Gonzales.

Management of the Failing Heart (Varick Pharmacal Company, Inc.)—Karnes-Wilson Counties Medical Society, Karnes City.

Mastoid Surgery (Dr. Louis Daily)—Scott and White Memorial Hospitals School of Nursing, Temple.

Megacolon, Total Colectomy with Ileoproctostomy for Hirschsprung's Disease (Dr. Philip Thorek)—Drs. Hyslop and Hyslop, Del Rio.

Middletown Goes to War (Texas Tuberculosis Association)—Scott and White Memorial Hospitals School of Nursing, Temple.

Obstructive Laryngitis (Mead Johnson)—Scott and White Memorial Hospitals School of Nursing, Temple, and Drs. Hyslop and Hyslop, Del Rio.

On the Firing Line (Texas Tuberculosis Association)—Scott and White Memorial Hospitals School of Nursing, Temple.

Once Upon A Time (Metropolitan Life Insurance Company)—Lions Club, Stratford.

Polio—Diagnosis and Management (British Information Services)—Graduate Registered Nurses Association, Cameron.

Pyloric Stenosis, Congenital, Surgical Treatment for (Dr. Philip Thorek)—Anderson-Houston-Leon Counties Medical Society, Palestine.

Question in Time, A (American Cancer Society)—Lions Club, Stratford.

Techniques of Injection (Becton, Dickinson and Company)—Hopkins County Memorial Hospital, Sulphur Springs.

They Also Serve (American Medical Association)—Drs. Hyslop and Hyslop, Del Rio.

They Do Come Back (Texas Tuberculosis Association)—Elizabeth Nixson Junior High School, Amarillo.

Traitor Within, The (American Cancer Society)—Scott and White Memorial Hospitals School of Nursing, Temple.

Transthoracic Esophageal Diverticulectomy (Dr. Philip Thorek)—Drs. Hyslop and Hyslop, Del Rio.

Urologic Conditions, Diagnosis of (Winthrop Chemical Company)—Hopkins County Memorial Hospital, Sulphur Springs.

Vagotomy for Ulcerative Colitis (Dr. Philip Thorek)—Anderson-Houston-Leon Counties Medical Society, Palestine.

What Is Cancer? (American Cancer Society)—Hopkins County Memorial Hospital, Sulphur Springs.

When Bobby Goes To School (Mead Johnson)—Kiwanis Club, Huntsville.

You Can Help (Texas Tuberculosis Association)—Drs. Hyslop and Hyslop, Del Rio.

BOOK NOTICES

[1]Tumors of the Head and Neck

Grant E. Ward, M. D., D. Sc., F.A.C.S., and James W. Hendrick, M. D., M. S.; from the Departments of Surgery of the School of Medicine, University of Maryland, and the Johns Hopkins University School of Medicine, and the Oncology Clinic, University Hospital and the Tumor Clinic of the Johns Hopkins Hospital. 832 pages, cloth. $15. Baltimore, Williams and Wilkins Company, 1950.

"Comprehensive" is perhaps the best term to apply to this book. The authors present an orderly description of tumors (benign, premalignant, and malignant) of the skin, lips, oral cavity, jaws, salivary tissue, tonsils, pharynx, larynx, paranasal sinuses, eye, ear, lymph tissues, skull, and other primary and secondary tumors of the head and neck. Thyroid tumors are included.

The etiology, clinical course, and treatment of these lesions are described in greatest detail. This arrangement makes the book of greatest value to the clinician and x-ray therapist. However, numerous photomicrographs of the lesions also are included, and these illustrations should appeal to pathologists.

This book deserves to be a standard reference for all branches of medicine.

[2]Atlas of Human Anatomy

M. W. Woerdeman, M. D., F.R.N.A. Sc., Professor of Anatomy and Embryology and Director of the Department of Anatomy, University of Amsterdam, Vol. 2, 673 pages, cloth. $10. Philadelphia, Blakiston Co., 1950.

This volume depicts the digestive, respiratory, urogenital, circulatory, nervous systems, and sensory organs. Drawings are chiefly in black and white and are reproduced with excellence. More than the usual amount of space is devoted to the teeth, with pictures of individual teeth in all planes. The system of nomenclature adopted by the German Anatomical Society at the meeting in Basel in 1895 (BNA) has been employed throughout.

[1]*Lloyd R. Hershberger, M. D., San Angelo.*
[2]*W. D. Roberts, M. D., Austin.*

Medical and dental students and physicians will find this volume a welcome addition to the library of anatomy.

[3]Primary Carcinoma of the Vagina

Robert G. Livingstone, M. D., Fabrikoid, 73 pages. $2. Springfield, Chas. C. Thomas, 1950. 1st edition.

This book gives an excellent outline of the location of carcinoma of the vagina with an excellent histopathological description.

For a book of this scope the outline of treatment is exceedingly hazy and leaves one in doubt as to the methods used in radium implantation and deep x-ray therapy. Also, the extent of surgical treatment is not well defined for adequate comparison of treatment.

[4]Acute Head Injuries

Joseph P. Evans, M. D., Associate Professor of Surgery, Director, Division of Neurological Surgery, University of Cincinnati College of Medicine, Cincinnati, Ohio. Fabrikoid, 116 pages. $2.25. Springfield, Ill., Charles C. Thomas, 1950.

A short chapter devoted to scalp wounds would seem rather elementary except for medical students and interns, whereas a subsequent chapter on skull fractures would concern the neurosurgeon only.

The discourse on meningeal hemorrhage is good. The author urges exploration in doubtful cases on the basis that a negative exploration is much less dangerous than a clot which is overlooked. Several pages are devoted to chronic subdural hematoma, emphasizing this possibility in any patient developing central nervous system signs after trauma.

The author makes a somewhat arbitrary classification of cerebral trauma based on the pathology of the various degrees of injury without making any clear clinical application.

The chapter on treatment of acute head injury is general and does not offer many useful points for the general practitioner.

This monograph represents a fine work, particularly as regards the pathology of acute head injury; however, those who are looking especially for points in diagnoses and management will be disappointed.

[5]Professional Adjustments

Sister Mary Isidore Lennon, R.S.M., R. N., M. A., Director of St. John's Hospital School of Nursing, 1939-1945; Director of Social Service Department, St. John's Hospital, St. Louis. Second edition. Cloth, $3.50. 362 pages. St. Louis, C. V. Mosby Company, 1950.

The second edition of this book by Sister Isidore contains the same fundamentals and principles as were set forth in the earlier edition, and the changes made have to do only with bringing the book up to date in regard to current economic and professional developments.

The ideals held up and the suggestions made to the student nurses are presented in an inspiring and stimulating manner, and the whole tone of the book is on an elevated plane. The book has been written primarily for Catholic nursing school libraries; this point of view is unfortunate as the material is excellent and could be of great value in the teaching of non-Catholic students of nursing. The principles outlined would benefit any group of students of whatever faith and would instill into them a deep sense of responsibility and an appreciation of the physical and spiritual needs of the patient.

The author has covered a vast amount of material in a single volume. The earlier units of the book are designed to present to the entering student principles of conduct and ideals toward which she should strive in her relationships with her companions, her patients, and the personnel of the hospital. The later units are concerned with problems and information that would be of value to the senior student, who will shortly be finding her way into the professional field. This portion of the book does not present enough detail to be offered to the student as a complete treatment of such material and yet it is too far-reaching for the young student who is just being introduced to the profession.

This book should be beneficial to the nurse administrator or the instructor in planning and teaching the early and later courses in professional adjustments, usually referred to as Professional Adjustments I and Professional Adjustments II. The objective type tests offered at the end of each chapter and also the suggested project topics should be helpful and time-saving for the teacher.

[6]Unipolar Lead Electrocardiography

Emanuel Goldberger, B. S., M. D., Adjunct Physician, Montefiore Hospital, New York; Cardiographer and Associate Physician, Lincoln Hospital, New York; Consulting Cardiologist, St. Joseph's Hospital, Yonkers, New York; Diplomate of the American Board of Internal Medicine; Lecturer in Medicine, Columbia University. 392 pages, cloth. $7.50. Philadelphia, Lea and Febiger, 1949.

This interesting book has been received somewhat coolly by some reviewers. In his persistent presentation of his views on the importance of positional changes and the necessity for unipolar leads, Dr. Goldberger has stepped on some toes. It must be said that the book, despite its prospects, devotes the absolute minimum of text to correlation with standard leads. The section on arrhythmias is largely unnecessary, especially as the book is in no way a complete text of electrocardiography.

However, the book is admirable in many ways, contains a great deal of readily available information compactly arranged, and obviously is written with care and sincerity by one of the recognized authorities in this field. It should have a place on every internist's book shelves.

[7]Language for the Preschool Deaf Child

Grace Harris Lassman, Teacher of the Deaf. Cloth, 263 pages. $5.50. New York, Grune and Stratton, 1950.

This book is a competent description of the general procedures available for the development of language communication in young deaf children. There are all too few such presentations to be had. As Harriet Andrews Montague, well-known educator of the deaf, says in a foreword, "American teachers of the deaf have been far too apt to follow Caroline Yale's famous observation that they were 'too busy doing the things to write about it.'" The author, drawing upon her experiences as a teacher of young deaf children at the John Tracy Clinic in Los Angeles, has written about the subject well. Her description of techniques, with careful attention to the *why* as well as the *how*, will do much to make the all-too-often inaccessible lore of the teacher of the deaf understandable and usable.

To the medical practitioner, so often the first competent person to whom the parent of the young deaf child comes for counsel and advice, this presentation will be particularly welcome. The many large illustrations help to carry a heartening message of what can be done for the child who does not hear to make him a happy and useful member of society.

It is to be hoped that many pediatricians, otologists, and general practitioners will familiarize themselves with this book and recommend it to parents of young deaf children.

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[3]*James H. Herrod, M. D., Austin.*
[4]*Mack F. Bowyer, M. D., Abilene.*
[5]*Sister Basil, Austin.*
[6]*Wylie F. Creel, M. D., Austin.*
[7]*Jesse J. Villarreal, Ph. D., Austin.*

ORGANIZATION SECTION

AMERICAN MEDICAL ASSOCIATION

Rural Health Conference

More than 700 farm and health leaders are expected to attend the sixth annual National Conference on Rural Health from February 23 to 24 at the Peabody Hotel, Memphis. It will be sponsored by the Committee on Rural Health of the American Medical Association in cooperation with national farm organizations.

"Why Wait—Let's Do It Ourselves" will be the theme of the program. Dr. F. S. Crockett, Lafayette, Ind., chairman of the Committee, will outline the national rural health program. The program this year will stress the planning and organization of community health councils.

State rural health committee chairmen will hold a preliminary meeting February 22, the day before the conference opens. The meeting will deal particularly with rural health problems from the physician's viewpoint.

Dr. Allen T. Stewart, Lubbock, regional director of the Committee on Rural Health of the A.M.A. and President-Elect of the State Medical Association, will preside at one session and will participate in a symposium on what can be accomplished by various organizations at the community levels. Another speaker will be Mr. J. Walter Hammond, Waco, President of the Texas Farm Bureau Federation.

Physicians who wish to attend should make reservations with Dr. Crockett at 535 North Dearborn Street, Chicago 10, and should make hotel reservations direct to the Peabody Hotel, Memphis.

STATE MEDICAL ASSOCIATION

Executive Council Meeting Reset

The meeting of the Executive Council which was scheduled for January 27 will be recessed until February 4 because all hotel rooms previously had been reserved for a convention in Austin on the date formerly set. The Council will convene at 9 a. m. in the Ballroom of the Stephen F. Austin Hotel. On February 3 at 6:30 p. m. a dinner meeting will be held in the Ballroom to which 1951 presidents and secretaries of county medical societies are invited. Also, meetings of boards, councils, and committees have been scheduled for February 3, and chairmen of these groups will be expected to give reports the following day at the Executive Council meeting. Chairmen who wish to meet prior to the Executive Council meeting should notify the central office of their requirements so that necessary arrangements can be made.

Deposit Needed for Annual Session Rooms

A deposit of $5 per day to hold any room reservation for the period of the State Medical Association annual session has been requested by the hotels and tourist courts of Galveston, Dr. E. S. McLarty, chairman of the Hotels Committee, reports. The deposit will be payable to the hotel or court upon notice from the Hotels Committee that a reservation is being made.

Deposits will be refunded to all physicians who are called into military service or who must meet any other serious emergency. Others who wish the deposit returned must give notice to the hotel at least seventy-two hours prior to the meeting.

Dr. McLarty is urging members of the Association and Auxiliary, technical exhibitors, and others planning to attend the annual session to write him at once at 202 Medical Building, Galveston, for reservations, as plans are now completed and he is mailing confirmations. The main session will be held May 1-2 with a meeting of the House of Delegates on April 29 and meetings of related organizations on April 30.

New Policy to Call Medical Reservists

Under a new, uniform and simplified system civilian groups will guide the Department of Defense in determining the total number of reserve medical officers to be called to active duty at any one time. The National Health Resources Advisory Committee of the National Security Resources Board and the Armed Forces Medical Policy Council will share this responsibility. Dr. Richard Meiling, Chairman of the Council, will submit estimated requirements of the three services to the two bodies for their views; final decisions will be made by the Secretary of Defense.

State and local Selective Service Advisory Committees (procurement and assignment committees) under the direction of the National Health Resources Advisory Committee also will advise the Department which individual reservists should be deferred, the final decision again to be made by the Secretary of Defense. This system will not affect members of Organized Reserve units who have certified their availability for immediate call to duty.

Male physicians who had not reached their fiftieth birthday registered with their local Selective Service boards January 15 with the exception of members of the regular or reserve components of the armed services and those doctors who had registered under the October 16, 1950, regulations. Previous military service did not remove the obligation of registering.

BUILDING CONTRACT AWARDED

Contracts for construction of the headquarters and library building of the State Medical Association were awarded January 2 after a meeting in Fort Worth of the Board of Trustees and the Building Committee. The general contract was awarded to Yarborough Construction Company of Austin. Construction on the new building to be erected at Lamar Boulevard and Nineteenth Street in Austin will begin immediately unless new emergency regulations intervene.

OFFICIAL FAMILY CHANGES

Dr. Robert W. Kimbro, Cleburne, was appointed January 2 by the Board of Trustees as chairman of the Committee on Public Relations to succeed Dr. George A. Schenewerk, Dallas, who had resigned from the committee. Dr. Kimbro also will serve as chairman for Texas for the National Educational Campaign.

Drs. Ellen Furey, Beaumont; J. M. Moore, San Antonio; and C. T. Ashworth, Fort Worth, have been named to the Advisory Committee to the Texas Society of Medical Technologists by Dr. William M. Gambrell, Austin, President of the State Medical Association. Dr. Ashworth will serve as chairman.

COUNTY SOCIETIES

Angelina County Society

December 11, 1950

(Reported by W. D. Thames, Jr., Secretary)

Officers of Angelina County Medical Society for 1951 were elected at a meeting December 11 in Lufkin. They are as follows: A. E. Percy, president; Kenneth Arnett, vice-president; Bernard Hyman, Lufkin, secretary; T. A. Taylor, delegate; Joe S. Burch, alternate; and R. W. Taylor, censor.

Bastrop County Society

November 2, 1950

Elgin physicians were hosts for the quarterly meeting of the Bastrop County Medical Society held in Bastrop State Park on November 2. Drs. and Mesdames Cliff Price and Lang F. Holland of Austin were special guests as were the wives of members. Dr. Holland, head of the diabetic clinic at Brackenridge Hospital in Austin, discussed the new treatments now being used in the clinic. Eighteen persons attended.

The society decided to notify military authorities that because of the small number of doctors in the county in proportion to the population, no physicians could be spared to the armed forces if high health standards were to be maintained at home.

Bee-Live Oak-McMullen Counties Society

December 18, 1950

(Reported by John W. Reagan, Secretary)

Self Examination of the Breast (motion picture).

At a dinner meeting December 18 in Beeville the following officers of Bee-Live Oak-McMullen Counties Medical Society were elected: J. W. Edmondson, president; G. W. Sansom, George West, vice-president; J. W. Reagan, Beeville, secretary-treasurer; L. W. Kirkland, Beeville, delegate; D. W. Davis, Three Rivers, alternate; and C. M. Poff, Tuleta, T. B. Reagan, Beeville, and G. W. Sansom, George West, censors.

A procurement and assignment committee was appointed by the president as follows: L. W. Kirkland, chairman, G. W. Sansom, and C. M. Poff.

The movie entitled above was shown and approved for showing before various women's organizations.

Bexar County Society

October 5, 1950

(Reported by J. J. Hinchey, Secretary)

Hemorrhoidectomy (motion picture with sound)—Kennedy Milburn, San Antonio.

Dr. Milburn gave the above paper when Bexar County Medical Society met October 5 in San Antonio.

James H. Strauch and Herman S. Wigodsky were voted unanimously into the society as members by application. It was voted that all members delinquent in their dues as of October 1 be so notified and that hospitals in Bexar County also be notified that they are not members in good standing of the society.

Jack Partain was elected a member of the civil defense advisory committee. The proposed revision of the constitution and by-laws was discussed.

October 19, 1950

(Reported by J. J. Hinchey, Secretary)

Management of Congestion in the Female Pelvis—B. H. Passmore, San Antonio.

Members of the Bexar County Medical Society and its executive board met October 19, in San Antonio. The above scientific program was presented; J. W. Palmer was section chairman for the evening.

Sydney Schiffer reported the plans of the National Diabetes Society for Diabetes Week. The society endorsed the drive and asked the cooperation of members in detecting latent cases of diabetes.

H. Walter Eyermann, O. Roger Hollan, David R. Oliver, and James K. Stewart were elected to membership.

The revised constitution and by-laws was adopted with a few additional amendments being proposed.

November 2, 1950

(Reported by J. J. Hinchey, Secretary)

Rheumatoid Arthritis—B. A. Merrick, Dallas.

The Bexar County Medical Society and executive board met November 2 in San Antonio. R. E. Fisher introduced Dr. Merrick, who spoke on the above topic.

Sidney Stovall and William Johnson were granted military leave of absence.

A request for approval of a mass chest roentgenographic survey of Alamo Heights school children was read, and approval was granted. It was announced that the American Cancer Society's new film "Self-Examination of the Breast" is now available.

The amendments made to the constitution at the previous meeting were adopted. John L. Matthews presented amendments defining further the duties of the public and professional relations committee.

Brazoria County Society

December 7, 1950

(Reported by W. T. Galloway, Secretary)

Oral Tumors (lantern slides)—E. C. Hinds, Houston.

Members of the Brazoria County Medical Society and Woman's Auxiliary met in Freeport on December 7. After dinner John M. Laughlin, Sweeney, introduced the speaker, who is on the faculty of the University of Texas School of Dentistry.

Auxiliary members invited the society to attend a dance on December 16, an annual observance of "Doctor's Day" for the society.

Officers for 1951 were elected as follows: R. C. Miller, Lake Jackson, president; Carlos E. Fuste, Jr., Alvin, vice-president; Joe S. Montgomery, Angleton, secretary-treasurer; and R. M. McCary, Freeport, censor.

The procurement and assignment situation was discussed, and H. E. Merz, Alvin, proposed the resolution that in view of the present doctor-patient population ratio that no doctors were available for procurement and assignment to the armed forces. His motion was seconded by Gerald F. Perryman, Velasco, and unanimously adopted.

Cass-Marion Counties Society

November 15, 1950

Hosts to the November 15 meeting in Hughes Springs were the local members of the Cass-Marion Counties Medical Society. Auxiliary members also attended the dinner, and afterward a tour of the steel plant was made. Ten persons were present.

Collin County Society

December 11, 1950

(Reported by Charley E. Wysong, Secretary)

The following officers of Collin County Medical Society were elected at the December 11 meeting: John M. Hooper, McKinney, president; and Charley E. Wysong, McKinney, secretary (re-elected).

Dallas County Society

November 14, 1950

(Reported by W. W. Fowler, Dallas, Secretary)

Some Renal Mechanisms of Salt and Water Excretion—Charles H. Burnett, Dallas.
Medical Economic News—Mr. Millard J. Heath, Dallas.

Elliott Mendenhall, president of the Dallas County Medical Society, at the November 14 meeting in Dallas presented two guests, Gilbert Forbes, professor of pediatrics at Southwestern Medical School, and Sidney Burwell, research professor of clinical medicine at Harvard Medical School, Boston, the honor guest at a postgraduate course sponsored by the Dallas Southern Clinical Society.

Sam Shelburne presented Dr. Burnett who gave the above scientific program.

Five members by transfer and one member by application were elected to membership. The president commended George Jones, Jr., Frank Kidd, Jr., and M. M. Scurry for their leadership in the Community Chest campaign, in which the quota of the medical profession had been exceeded.

Donald Paulson, chairman of the committee on tuberculosis, discussed a survey proposed by the United States Public Health Service. He offered a resolution that the society in coordination with the Dallas Tuberculosis Association and the city and county health departments sponsor a chest x-ray survey among adults of the county; that the Texas State Department of Health be invited to assist by furnishing a suitable number of mobile units equipped with personnel, films, and records; and that the society in cooperation with the association and local health departments form an executive committee to make plans for the survey and follow-up study. The resolution was seconded by Dr. Paulson and adopted.

Tate Miller, chairman of the procurement and assignment committee, reported that the committee had been officially designated as an advisory committee to the Selective Service Board and that it was now functioning. He explained the committee's duties and asked the cooperation of society members.

The recommendation made June 13 that Negro physicians be invited to attend the scientific programs of the society was properly seconded and approved.

The society studied the proposed revision of the constitution and by-laws, but after considerable discussion the motion for adoption failed.

Ector-Midland-Martin-Howard-Andrews-Glasscock Counties Society

November 16, 1950

Curable Heart Diseases—John S. Bagwell, Dallas.

Dr. Bagwell presented the paper named above at the regular monthly meeting of Ector-Midland-Martin-Howard-Andrews-Glasscock Counties Medical Society in Midland on November 16.

December 14, 1950

(Reported by James W. Rainer, President)

Cancer of Colon (x-rays)—Alvin Baldwin, Jr., Dallas.

At a dinner meeting December 14 in Big Spring the following officers were elected to serve Ector-Midland-Martin-Howard-Andrews-Glasscock Counties Medical Society for 1951: C. S. Britt, Midland, president; Clyde E. Thomas, Big Spring, vice-president; and Lex Smith, Midland, secretary.

Dr. Baldwin, a Dallas proctologist, gave a paper "Cancer of the Colon."

Gonzales County Society

December 13, 1950

(Reported by James C. Price, Secretary)

A.M.A. Convention (motion picture newsreel).
Infant Diarrhea—James C. Price, Gonzales.

The above scientific program was presented at the December 13 meeting in Gonzales of the Gonzales County Medical Society. Officers for 1951 were elected as follows: James C. Price, president; D. A. Schramm, vice-president; L. J. Stahl, secretary; and N. A. Elder, delegate. Dr. Elder is of Nixon; the other officers from Gonzales.

Gray-Wheeler-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Society

November 21, 1950

(Reported by J. R. Donaldson, Pampa, Secretary)

Intestinal Obstruction in Childhood—A. O. Singleton, Jr., Galveston.
Chest Surgery in Childhood—A. W. Harrison, Galveston.

W. W. Brooks, Borger, first vice-president, presided at the November 21 meeting of Gray-Wheeler-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Medical Society in Pampa in the absence of the president. Twenty-five members and four guests attended.

Wayne Bagwell, Borger, introduced the speakers, who with their topics are given above. The question of 1950 A.M.A. dues was discussed and the secretary proposed an amendment to the constitution that dues be collected by the secretary-treasurer of the society, being included with the local and state dues payable by January of each year. The amendment was tabled until the next meeting.

A letter from Erath-Hood-Somervell Counties Medical Society regarding the transfer of Robert R. Surratt, now practicing in Pampa, and a note of thanks from the family of the late Dr. J. H. Kelley of Pampa were read.

Grayson County Society

November 14, 1950

(Reported by G. S. Rowlett, Jr., Secretary)

Heart Disease in Surgical and Obstetric Conditions—W. J. Gleckler, Sherman.

The paper named was presented at a dinner meeting of Grayson County Medical Society in Denison on November 14.

December 12, 1950

(Reported by G. S. Rowlett, Jr., Secretary)

Electrocardiography (motion picture).

A dinner meeting of the Grayson County Medical Society was held in Denison on December 12. In a business session officers for the new year were elected as follows: D. J. Donaghey, Sherman, president; Robert W. Duncan, Denison, vice-president; and G. S. Rowlett, Jr., Sherman, secretary (re-elected).

The above-named motion picture was shown.

Harris County Society

November 8, 1950

The methods and problems of setting up a regional blood bank were outlined by Russell Landram Haden, Cleveland, medical director of the American Red Cross national blood program, speaking before members of the Harris County Medical Society in Houston on November 8. A final decision on how to develop a blood bank in Houston is under consideration by the society.

Hill County Society

December 9, 1950

(Reported by Charles A. Garrett, Secretary)

Amputation Prosthesis (motion picture).

Eleven members of Hill County Medical Society met in

Hillsboro for dinner and a business session. The secretary reported that letters regarding the transfer of John W. Harper from Wichita County Medical Society had been written. Robert W. Shirey moved and N. C. Smith seconded that the Texas State Department of Health be requested to furnish the services of mobile x-ray units for a tuberculosis survey in Hill County; the motion carried.

The secretary read a financial report which was accepted by the society.

Officers for 1951 were elected. They are Dick Cason, president; Robert W. Shirey, vice-president; Charles A. Garrett, secretary-treasurer; Robert W. Shirey, delegate; T. R. Barnett, alternate (1950-1951); James M. Buie, censor. All are of Hillsboro except Dr. Buie who is of Whitney.

The president appointed committees for 1951. It was reported by the secretary that he had accepted appointment for three years on the public and professional relations committee for the Twelfth District.

Jefferson County Society

(Reported by E. Mittendorf, Executive Secretary)

The following physicians will serve as officers of Jefferson County Medical Society for 1951: Lamar C. Bevil, Beaumont, president; W. Price Killingsworth, Port Arthur, president-elect; John M. White, Jr., Port Arthur, vice-president; R. H. Engledow, Beaumont, secretary-treasurer; L. C. Heare, Port Arthur, and J. C. Crager, Beaumont, delegates; W. J. Graber, Beaumont, and L. C. Carter, Port Arthur, alternates; and W. A. Smith, Beaumont, R. C. Willoughby, Groves, and W. E. McRee, Jr., Port Arthur, censors.

Liberty-Chambers Counties Society

(Reported by Don P. Schulz, Secretary)

Newly elected officers of Liberty-Chambers Counties Medical Society are as follows: C. W. Castle, Anahuac, president; Melvin Anchell, Cleveland, vice-president; Reginald Wilson, Dayton, secretary-treasurer; R. C. Bellamy, Daisetta, D. P. Schulz, Liberty, and T. L. Fahring, Anahuac, censors; A. R. Shearer, Mont Belvieu, delegate; and G. H. Fahring, Anahuac, alternate.

Lubbock-Crosby Counties Society

November 7, 1950

(Reported by Frank W. Hudgins, Secretary)

Diagnosis of Primary Carcinoma of the Lung (slides)—H. F. Carman, Dallas.
Discussion—Richard Q. Lewis, Albert G. Barsh, and Frank B. Malone, Lubbock.

The scientific program outlined was presented when Lubbock-Crosby Counties Medical Society met November 7 in Lubbock. Approximately thirty members and guests were present for the program and business session. Upon motion by J. D. Donaldson, seconded by Chris C. Mansell, the society decided to sponsor a diabetes detection drive. A letter from the premedical adviser at Texas Technological College announced the names of two students chosen to receive premedical scholarships this year. A nominating committee was appointed, and a letter transferring the membership of Dr. Charles A. Pigford from Hale-Floyd-Briscoe-Swisher Counties Medical Society to Lubbock-Crosby Society was read.

December 5, 1950

(Reported by Frank W. Hudgins, Secretary)

Mortality and Morbidity in Appendicitis During the Last Fifteen Years—Ewell L. Hunt, Lubbock.
Discussion—Robert H. McCarty, Olan Key, J. H. Stiles, Otis W. English, Lubbock.

Approximately thirty-five members and guests attended the December 5 meeting in Lubbock of Lubbock-Crosby Counties Medical Society. Elected by acclamation to office for 1951 were the following: Mina D. Watkins, president; Ewell L. Hunt, vice-president; John B. Rountree, Jr., secretary-treasurer; Frank B. Malone, councilor; Sam C. Arnett, Jr., delegate; and Ivan G. Mayfield, Emerson M. Blake, and Myron D. Mattison, censors.

Ivan G. Mayfield reported on the recent tuberculosis and diabetes drives in Lubbock. The above scientific program was given.

McLennan County Society

November, 1950

New officers of McLennan County Medical Society elected at a November meeting are W. M. Avent, president; James Colgin, vice-president; C. C. Smith, secretary-treasurer; and B. F. Roach, censor.

Nueces County Society

December 12, 1950

(Reported by M. C. Kendrick, Secretary)

Infections of Skin and Fascia—Carl A. Moyer, Dallas.

At the December 12 meeting in Corpus Christi of Nueces County Medical Society officers were elected with the following results: Jerome Nast, president; F. W. Yeager, president-elect; Hugh A. Kennedy, vice-president; M. C. Kendrick, secretary; and Paul M. Gray, treasurer.

After Dr. Moyer's talk, the topic of which is stated above, a question and answer period was held.

Pecos-Jeff Davis-Presidio-Brewster Counties Society

November 7, 1950

(Reported by W. E. Lockhart, Secretary)

Cancer of Lung—Delphin von Briesen, El Paso.
Bronchoscopy, New Concepts and New Applications—Haskell Hatfield, El Paso.

Seven members of Pecos - Jeff Davis - Presidio - Brewster Counties Medical Society and three guests were present for a dinner meeting November 7 at the Gipson Hospital, Fort Stockton. J. F. Gipson was host for the meal. The scientific program outlined was presented by two guests from El Paso.

December 13, 1950

(Reported by W. E. Lockhart, Secretary)

Elected to office for 1951 in the Pecos-Jeff Davis-Presidio-Brewster Counties Medical Society were the following: V. A. Sherrod, Iraan, president; Malone V. Hill, Alpine, vice-president; D. J. Sibley, Jr., Fort Stockton, secretary; W. H. Stover, Marfa, treasurer; and John W. O'Donnell, Alpine, delegate.

Potter County Society

November 13, 1950

(Reported by Mrs. Cassie Atherton, Executive Secretary)

Potassium Metabolism; Its Importance in Medicine and Surgery—Andrew Small, Dallas.

Dr. Small, assistant professor of surgery at Southwestern Medical School, Dallas, was guest speaker when about fifty members of Potter County Medical Society met in Amarillo on November 13. A motion picture, "Highlights of Socialism," was presented by Mrs. Ralph Payne, member of the auxiliary.

The society voted to amend its by-laws to include American Medical Association dues in its annual assessment. William Wheir reported that a recent roentgen-ray survey to discover chest abnormalities resulted in 12,000 films taken by the State Health Department, a number greater than the total obtained in a former survey. John M. Pickett was accepted for membership upon transfer from Harris County. The society voted to place an advertisement in the high school annual.

December 11, 1950

(Reported by Mrs. Cassie Atherton, Executive Secretary)

A gold headed cane was presented to Howard E. Puckett, incoming president of the Potter County Medical Society, by outgoing President Waddill at the December 11 meeting in Amarillo. First presentation of the cane was made in 1905 to the late Dr. W. A. Lockett, who requested that it be returned to the society after his death and handed on to each incoming president.

Other officers were elected at the meeting as follows: Gaylord R. Chase, secretary-treasurer (re-elected); T. P. Churchill, delegate; Fred Crumley, alternate; George Powers, George Waddill, and William R. Klingensmith, censors; and Capres S. Hatchett, editor of the society's *Bulletin*.

About fifty members were present for the meeting. Visitors were Mrs. Rhea Smith and Mrs. Warren Reeves of the Potter County chapter of the American Red Cross.

T. P. Churchill reported on the blood bank and was commended for his work during the past year. A report for the heart committee was made by H. H. Latson and Francis Kelly. Ben T. Blackwell reported for the procurement and assignment committee.

Secretary Gaylord R. Chase made a financial report for the year. A motion that the president appoint from three to five members to work with H. H. Latson on the civil defense program was seconded and carried.

Tarrant County Society

November 7, 1950

(Reported by W. P. Higgins, Jr., Secretary)

Ideal Biopsy—John J. Andujar, Fort Worth.
Symposium on Lung Tumors—C. T. Ashworth, John A. Wiggins, and Eugene D. Olcott, Fort Worth.

Seventy-two members of Tarrant County Medical Society heard the program outlined when they met November 7 in Fort Worth. Plans for the local diabetes detection drive were announced, and the president-elect, Joseph F. McVeigh, discussed some suggestions for the 1951 activities of the society. Mal Rumph presented a proposed amendment to the by-laws to provide for inclusion of American Medical Association dues in the annual dues of the society.

Roy E. Snyder was elected to membership.

November 21, 1950

(Reported by W. P. Higgins, Jr., Secretary)

Melanomas—Harold J. Shelley, Fort Worth.

Seventy-nine members attended the meeting November 21 in Fort Worth of Tarrant County Medical Society.

Bernard Schnitzer was elected to membership. Tribute was paid to Dr. F. H. Anthony who died November 5.

An amendment regarding local, state, and national dues was adopted. T. C. Terrell and R. G. Baker discussed the A.M.A. dues for 1950 and urged members to pay this fee.

The president introduced Walter Humbert, medical director of the defense blood program of the American Red Cross. The society voted its endorsement of the program and the president was authorized to appoint a committee to form plans and make necessary agreements, subject to ratification by the society.

Bert Ball reported for J. D. Murphy, a member of the committee on diabetes, that during Diabetes Detection Week, 7,963 tests were made. Dr. Ball also reported the nominees which had been chosen by the nominating committee.

R. G. Baker moved that the legislative committee instruct members to write the members of the Legislature regarding the Licensure Bill.

December 5, 1950

(Reported by W. P. Higgins, Jr., Secretary)

Tarrant County Medical Society members met in Fort Worth on December 5 with 150 members present. Annual reports were given by the various officers and committees. Officers for 1951 were elected as follows: Hub E. Isaacks, president-elect; Robert D. Bickel, vice-president; W. P. Higgins, Jr., secretary-treasurer; George Lacy, censor; May Owen, trustee; W. B. West and E. P. Hall, Jr., delegates; and W. F. Armstrong and Mal Rumph, alternates. Dr. Joseph F. McVeigh will serve as president in 1951.

Tribute was paid Dr. F. W. Francis who died December 2, 1950. Tom B. Bond moved and W. G. Phillips seconded that Ross Trigg be nominated for honorary membership in the State Medical Association; the motion carried. The second meeting in December was dispensed with because of the Christmas holidays.

Tom Green-Eight County Society

November 6, 1950

(Reported by H. M. Anderson)

Current Problems Facing Blue Cross and Blue Shield Insurance Companies in Texas—W. R. McBee, Dallas.
Advantages and Disadvantages of Prepaid Hospitalization for Rural People—C. H. DeVaney, Coahoma.

Thirty-two persons were at the meeting November 6 in San Angelo of Tom Green-Eight County Medical Society. The nominating committee reported its selection of a slate of officers for 1950.

A letter regarding the transfer of membership of Marion L. Wood was read. Reports were given by Lloyd Hershberger for the athletic committee and by Harvey Williams for the military committee.

The program outlined above was given. After the meeting society members met the members of the auxiliary for a social hour.

December 5, 1950

(Reported by Perry J. C. Byars, Jr., Secretary)

A large number of local druggists met for dinner with thirty-nine members of Tom Green-Eight County Medical Society in San Angelo on December 4 after a cocktail party given by the druggists.

The president, Carl Kunath, reported the latest developments of the indigent care program being established in the county and city. Lloyd Hershberger commented on some of the recent problems of the cancer committee, and R. E. Windham, Fourth District Councilor, gave a report supplementing Dr. Hershberger's comments.

William J. Swann, Sterling City, moved and H. M. Anderson seconded that $5 be assessed to cover the deficit shown in the financial statement. The motion carried unanimously.

A letter of transfer of Marthalyn Gainer from Galveston County Medical Society was read.

Officers who will serve the society in 1951 are as follows: M. D. Knight, president; Perry J. C. Byars, president-elect; R. M. Arledge, vice-president; Gordon Madding, secretary; W. D. Anderson, censor; James N. White, delegate (re-elected), and Henry Ricci, alternate (re-elected).

Dr. Kunath gave his retiring presidential address and Dr. Knight made a short acceptance talk.

Travis County Society

October 17, 1950

(Reported by John F. Thomas, Secretary)

A newsletter was authorized at the October 17 meeting in Austin of Travis County Medical Society. It was decided that four meetings a year will be devoted entirely to business, the other six being wholly scientific. New members of the society are Robert N. Bugg, Richard A. Lucas, and Charles E. McKenzie.

November 21, 1950

(Reported by John F. Thomas, Secretary)

If Disaster Comes (motion picture)—Col. Victor Byrnes, School of Aviation Medicine, Randolph Field.

Colonel Byrnes outlined the effects of the atomic bomb from a medical standpoint, particularly from the ophthalmologist's viewpoint, at the meeting in Austin on November 21 of Travis County Medical Society.

Webb-Zapata-Jim Hogg Counties Society

December 5, 1950

(Reported by Aubrey S. McGee, Secretary)

Officers for 1951 were elected at the meeting December 5 of Webb-Zapata-Jim Hogg Counties Medical Society. They are as follows: W. R. Powell, president; Max Rottenstein, vice-president; and A. S. McGee, secretary-treasurer. All officers are of Laredo.

Wharton-Jackson-Matagorda-Fort Bend Counties Society

(Reported by J. J. Hollomon, Jr., Secretary)

The following have been named as officers for 1951 of the Wharton-Jackson-Matagorda-Fort Bend Counties Medical Society: J. M. Bauknight, Ganado, president; Howard Z. Fretz, Wharton, vice-president; S. R. Mortland, Ganado, secretary-treasurer; W. S. Thiltgen, El Campo, chairman, and S. R. Mortland, Ganado, and C. A. Shoultz, Bay City, censors; H. C. Matthes, Bay City, and V. A. Black, Wharton, members of the board of adjudication; R. D. Zipp, Edna, and R. D. Little, Wharton, executive board members; L. B. Johnson, El Campo, delegate; Harold Northington, Wharton, and J. N. Bader, Edna, alternates.

Williamson County Society

December 12, 1950

Officers of Williamson County Medical Society who were elected December 12 are as follows: Dewey H. Cooper, Georgetown, president; H. R. Gaddy, Jr., Georgetown, vice-president; John C. Godbey, Jr., Taylor, secretary-treasurer; Albert J. Rice, Georgetown, delegate; and Dick B. Gregg, Round Rock, alternate.

DISTRICT SOCIETIES

Fourth District Society

November 1, 1950

(Reported by S. Braswell Locker, Secretary)

Choice of Fluids in Medical Care—Merton M. Minter, San Antonio.
Choice of Fluids in Surgical Care—H. Vincent Walker, San Antonio.
Glaucoma and Its Interest to the General Practitioner—Lester H. Quinn, Dallas.
Nasal Allergy and Chronic Sinusitis—James T. Hall, Lubbock.
Problem of Anuria and Oliguria—M. K. O'Heeron, Houston.
Surgical Conditions of Gastrointestinal Tract in Children—A. O. Singleton, Jr., Galveston.
Diagnosis and Management of Lung Abscess—J. W. Middleton, Galveston.
Breast Tumors—Jarrett Williams, Abilene.

The scientific program outlined above was given at the meeting in Brownwood on November 1 of the Fourth District Medical Society which was attended by about fifty physicians. The district auxiliary had a luncheon and program on the same date.

Principal speaker at the banquet in the evening was William M. Gambrell, Austin, President of the State Medical Association.

Officers elected for 1951 were S. Braswell Locker, Brownwood, president; James B. N. Walker, Brownwood, vice-president; and B. A. Hallum, Brady, secretary-treasurer.

The society will meet again in November, 1951, in Brady.

Ninth District Society

November 11, 1950

(Reported by J. T. Billups, Councilor)

An organizational meeting of the Ninth District Medical Society was held November 11 in Houston with J. T. Billups, councilor of the district, presiding. Officers were elected as follows: L. E. Bush, Huntsville, president; O. F. Schoenvogel, Brenham, vice-president; and Lyman C. Blair, Houston, secretary-treasurer. It was suggested that the president appoint a committee to submit a constitution and by-laws to the society for approval and a committee to work out a district meeting, probably for February, 1951.

AUXILIARY SECTION

STATE PRESIDENT CONTINUES VISITS

Mrs. William M. Gambrell, Austin, President of the Woman's Auxiliary to the State Medical Association, expects to continue her visits to county and district auxiliaries through March and has expressed her willingness to include additional cities in her itinerary if she receives invitations soon.

Mrs. Gambrell also reminds that the program for the Auxiliary's annual session, to be held in Galveston from April 29 to May 2, will be published in the March issue of the JOURNAL.

AUXILIARY NEWS

Angelina County Auxiliary

The Angelina County Auxiliary met October 31 with twelve members attending. Mrs. Joe Burch, president, and Mrs. W. D. Thames, Jr., of Lufkin were co-hostesses.

Mrs. P. C. Clements, Lufkin, was hostess for the November 28 meeting at which Mrs. T. A. Taylor gave a report of the Postgraduate Medical Assembly of South Texas, which met in Houston recently. Mrs. Bertram Menefee told the story "Mama Entertains." Ten members were present.—Mrs. W. D. Thames, Jr., Secretary.

Bell County Auxiliary

Dr. A. Ford Wolf, chairman of the Bell County Medical Society's advisory committee to the auxiliary, spoke at the first meeting of the auxiliary in October in Temple. He was introduced by Mrs. Raleigh Curtis, president.

Mrs. Frank Covert introduced six new members and members of the Bell County Auxiliary who are on the executive board of the state auxiliary: Mrs. V. M. Longmire, finance committee; Mrs. A. E. Moon, memorial service; and Mrs. G. V. Brindley, Sr., special advisor to the state president.

Officers of the Woman's Auxiliary to the State Medical Association of Texas: President, Mrs. William M. Gambrell, Austin; President-Elect, Mrs. O. W. Robinson, Paris; First Vice-President (Organization), Mrs. J. H. McCracken, Dallas; Second Vice-President (Physical Examinations), Mrs. Allen T. Stewart, Lubbock; Third Vice-President (Today's Health), Mrs. Joe T. Nichols, Atlanta; Fourth Vice-President (Program), Mrs. Carlos Hamilton, Houston; Recording Secretary, Mrs. E. W. Coyle, San Antonio; Treasurer, Mrs. John D. Gleckler, Denison; Corresponding Secretary, Mrs. William P. Morgan, Austin; Publicity Secretary, Mrs. R. T. Wilson, Austin; Parliamentarian, Mrs. Robert F. Thompson, El Paso.

Two out-of-town guests attended. A skit "Rehearsal for Duty" was presented by the incoming officers. Refreshments were served by the hostesses, Mesdames David Eanes, H. B. Anderson, A. H. Alsup, R. N. Bartels, P. M. Bassel, E. O. Bradfield, G. V. Brindley, Sr., John J. Christian, John M. Coleman, and Frank Covert.

Speaker for the meeting November 10 in Temple was Mrs. William M. Gambrell, Austin. She, Mrs. Curtis, president of the local auxiliary, and two past state presidents, Mrs. F. F. Kirby and Mrs. Howard Dudgeon, Sr., both of Waco, and Mrs. L. D. Stuart, Bryan, president of Brazos-Robertson Counties Auxiliary, were honored at a luncheon.

Mrs. Brindley, leader of the program, introduced Mrs. Gambrell. Mrs. Paul Ramey gave the invocation.

The auxiliary voted to send $2 to the National Federation of Women's Club's convention fund, to invite a home demonstration president as guest of the December Federation luncheon, and to sponsor a needy family during the Christmas season.

In charge of arrangements were Mesdames L. M. Cochran, J. W. Althaus, D. M. Bornstein, H. M. Bair, R. E. Pleune, Herbert Schubert, T. K. Young, and A. T. Hume.

Bexar County Auxiliary

Members of the Woman's Auxiliary to Bexar County Medical Society met with members of the bar auxiliary on November 27 in the home of Mrs. Ernest W. Clemens, San Antonio. Mrs. George M. Luhn was co-hostess.

Dr. Boen Swinny, who returned recently from England, spoke on his impressions of socialized medicine in Great Britain. Special guests were Mrs. Swinny, Mrs. Charles L. McGehee, and Mrs. A. O. Severance. Presiding at the table were Mesdames J. C. Hall. Theo F. Weiss, Arley V. Knight, and George H. Spencer.

A short business meeting was held December 8 in San Antonio. On December 9 the auxiliary entertained with a formal Christmas dinner dance in the Minuet Room of the Menger Hotel. Mesdames John Parsons, social chairman, and Charles McGehee, president, greeted guests, assisted by Mesdames J. J. DeLeon and Robert Gossett, co-chairmen.

Christmas decorations were used, and a buffet supper was served.—Mrs. Brad Oxford, Publicity Secretary.

Cherokee County Auxiliary

The annual Thanksgiving meeting and banquet of the Cherokee County Medical Society and Auxiliary was held November 29 in Rusk with more than 100 guests attending. A musical program "An Old Fashioned Garden" was given by patients of the Rusk State Hospital under the direction of Mrs. Essie Madden, recreational director of the hospital. The menu and decorations were planned by Mrs. Ester Mai Durrett, hospital dietitian.

Denton County Auxiliary

Mrs. Dora Foster, Dallas, executive director of the Richmond Freeman Memorial Clinic, talked on the organization of hospitals and hospital auxiliaries at a recent public meeting sponsored by the Denton County Auxiliary. The meeting followed a luncheon for auxiliary members and their guests. During a business meeting four new members were voted into the auxiliary.

Galveston County Auxiliary

Wives of newcomers to the faculty of the University of Texas Medical Branch Medical Dames, and women medical students were honored at the annual tea given in the home of Mrs. W. J. Jinkins, Jr., Galveston, on November 14 by the Galveston County Auxiliary.

In the receiving line were Mrs. John Middleton, president of the auxiliary; Mrs. E. L. Lancaster, president of the Women Medical Students' organization; Mrs. Donald Duncan, president of the Women's Faculty Club; and Mrs. Edward M. Shapiro, president of the Medical Dames.

Mrs. W. J. Jinkins, Sr., was in charge of decorations. Presiding at the silver services were Mesdames W. F. Spiller, George T. Lee, Chauncey D. Leake, E. L. Porter, G. W. N. Eggers, W. J. Jinkins, Jr., Clarence Sykes, and Arild Hansen.

Mrs. Y. C. Smith, Jr., harpist, played background music during the tea. Co-chairmen of arrangements were Mrs. A. O. Singleton, Jr., and Mrs. W. C. Levin, and hostesses were Mesdames William H. Ainsworth, Edward D. Futch III, Norman D. Jarrell, J. L. Jinkins, C. C. Morris, Theo Panos, and John Varner.

Gregg County Auxiliary

The Gregg County Auxiliary met recently in Kilgore and heard an address by Mrs. W. M. Gambrell, Austin, president of the Woman's Auxiliary to the State Medical Association. Twenty-four members were present.

Harris County Auxiliary

The annual Christmas dinner-dance of the Harris County Auxiliary was held at the River Oaks Country Club, Houston, on December 2. New members and their husbands were introduced. Bob Sheehan's orchestra played.—Mrs. Elizabeth Zarr, Publicity Chairman.

Hidalgo-Starr Counties Auxiliary

The Woman's Auxiliary to Hidalgo-Starr Counties Medical Society were hosts to their husbands at a dinner meeting of the two groups November 9 in Mercedes. Dr. R. H. Johnston played piano selections, and Miss Thelma Cade sang, accompanied by Miss Virginia Nylander.

Nacogdoches County Auxiliary

Approximately 200 guests attended the fourth annual silver tea of the Nacogdoches County Auxiliary in the home of Dr. and Mrs. James G. Taylor, Nacogdoches, on November 15. Funds went toward the furnishing of a room in the wing under construction at City Memorial Hospital, Nacogdoches.

Miss Sally Taylor, daughter of the hostess, and Miss Ellen Beall, daughter of Dr. and Mrs. J. Frank Beall, greeted the guests. Mrs. Clarence Smith registered them. In the house party were Mesdames Stephen B. Tucker, Walter B. Allen, J. Frank Beall, C. F. Neuville, and A. L. Nelson.

Assisting in serving were Mesdames George Barham, Sr., A. L. Nelson, T. J. Pennington, and G. F. Middlebrook, and Misses Elizabeth Burk, Harriet McKinney, Elizabeth Monk, and Sarah Alice Gilmore.—Mrs. A. L. Nelson.

Potter County Auxiliary

"Doctor's Day" was observed November 14 by Potter County Auxiliary at a luncheon meeting in Amarillo. Auxiliary members also sent their husbands red carnations in celebration and provided speakers on medical subjects to a number of civic clubs. Mrs. Ralph Payne presented a motion picture on the pros and cons of socialism. Hostesses for the luncheon included Mesdames Frank Duncan, Jan Werner, Ben Blackwell, R. D. Gist, Peter Garre, and Robert Gleason. —Mrs. Howard Reid, Secretary.

Smith County Auxiliary

Co-hostesses for the recent meeting and coffee attended by thirty-six members in Tyler of the Smith County Auxiliary were Mrs. Ben M. Wilson, Mrs. Henry Poetter, and Mrs. P. DeCharles. The program "Free Health Services in Our Community" was presented by Mrs. R. E. Baldwin and Mrs. Hascall Muntz. Mrs. Harold Cameron was program chairman. Mrs. Weldon Birdwell, president, was in charge of the meeting.

Mrs. Ed Faber spoke on the Tyler League of Women

Voters and extended an invitation to members of the auxiliary to join.

Tarrant County Auxiliary

Mrs. Mal Rumph, president, and new members were honored by Tarrant County Auxiliary at its first meeting of the year October 13 in Fort Worth. Mrs. G. Herbert Beavers, Jr., spoke on "Knowing Fort Worth." Hostesses for the luncheon were Mesdames Charles H. McCollum, Jr., and S. J. R. Murchison.

A public relations meeting of 327 women representing approximately 100 civic, educational, and social organizations of Fort Worth and nearby communities was held November 10 in Fort Worth by Tarrant County Auxiliary. Howard M. Fender, Fort Worth attorney, spoke on "This Is Our Problem," outlining the history of socialism, its encroachment in the United States, the superiority of "the American Way of Life," and the necessity of maintaining that way of life. Literature and resolutions pertinent to the subject were distributed. Mr. Fender was introduced by Mrs. Drew O. D. Ware, first vice-president and program director for the day. Mrs. Truman C. Terrell, public relations chairman for the State Auxiliary; Mrs. Edwin C. Davis; and Mrs. Hobart Deaton presided at the coffee services during a social period.—Mrs. Jack E. Daly, Publicity Secretary.

Taylor-Jones Counties Auxiliary

Mrs. William M. Gambrell, Austin, President of the Woman's Auxiliary to the State Medical Association, spoke on the influence of doctors' wives in molding sentiment in America against compulsory health insurance at a meeting of Taylor-Jones Counties Auxiliary on October 3 in Abilene. Mrs. Donald McDonald, president, introduced the speaker.

Dr. L. J. Webster and Dr. Travis Smith outlined the local plans for advertising the National Education Campaign of the American Medical Association during October. Dr. Webster discussed projects of the medical society, which include the establishment of a free clinic for indigents and medical indigents, a school for practical nurses, and a doctors' call service.

Mrs. Gambrell was the guest of honor at a dinner given in the home of Mrs. L. J. Pickard. Hostesses for the dinner with Mrs. Pickard were Mesdames Jack F. Bowyer, Jack Crow, Guy Pattillo, and Bernard H. Ailts.

Travis County Auxiliary

Members of the Travis County Auxiliary and their guests were entertained with a coffee November 14 in Austin. Guest speaker was Dr. William M. Gambrell, Austin, President of the State Medical Association. Reports were given by Mrs. T. J. Archer, budget committee; Mrs. Allen Neighbors, Jr., community service; Mrs. Sam Wilborn, *Today's Health*; Mrs. J. R. Nichols, courtesy committee; Mrs. George F. Tipton, health education; and Mrs. G. W. Cleveland, legislative committee.

Mrs. Gambrell, president of the Woman's Auxiliary to the State Medical Association, officiated at the coffee service.

Eleventh District Auxiliary

Mrs. R. T. Travis, president of the Cherokee County Auxiliary, was guest speaker in the absence of Mrs. O. W. Robinson, Paris, President-Elect, at a meeting of the Eleventh District Auxiliary in Jacksonville October 26.

An informal coke party was held after the business meeting, and Mrs. Thomas Jarmon of Tyler and her hospitality committee were responsible for the refreshments. The auxiliary met with members of the Eleventh District Medical Society for dinner in the evening, at which Dr. J. M. Travis, Jacksonville, introduced Dr. William M. Gambrell, Austin, President of the State Medical Association, as guest speaker.

DEATHS

T. B. TAYLOR

Dr. Thomas Bell Taylor, Jourdanton, Texas, died in Pleasanton on October 21, 1950, from primary carcinoma of the lung.

Born in Lexington, Tenn., on September 1, 1862, Dr. Taylor was the son of Thomas L. and Mary (Andrews) Taylor. He acquired his academic education in Tennessee and in 1886 was graduated from the Medical Department of Tulane University of Louisiana, New Orleans. Dr. Taylor began his medical career in Elgin. He served a residency in surgery in 1909 and took postgraduate work in Chicago. He then was located in Olive, Muldoon, and Paige for about eight years before moving to Bastrop, remaining there for fifty years. Eight years ago he moved to Jourdanton and practiced until four weeks before his death, being the oldest living graduate of Tulane University medical school in active practice.

Dr. Taylor was a member of the State Medical Association and the American Medical Association through Bastrop County and Atascosa County Medical Societies. In 1948 he was named to honorary membership in the Association. For sixteen years he was county health officer for Bastrop County; also, he formerly was city health officer of Jourdanton. Dr. Taylor while in Bastrop was a surgeon for the Missouri-Kansas-Texas Railroad. He was an elder in the Presbyterian Church for more than sixty years and was chairman of the Bastrop County chapter of the American Red Cross.

On September 24, 1887, in Tennessee Dr. Taylor married Miss Pauline Fulton, who survives. Also surviving are his son, Thomas Bell Taylor, Jr., D.D.S., Houston; three daughters, Mrs. H. G. Lytton, Baldwin Park, Calif., Mrs. J. F. Metcalfe, Elgin, and Mrs. Jim Denison, Pleasanton; eight grandchildren; and four great-grandchildren.

G. W. WORTHINGTON

Dr. Glover W. Worthington, Marathon, Texas, died November 9, 1950, in Alpine from an aortic aneurysm.

Dr. Worthington was born June 8, 1874, in Lovelady, the son of James M. and Willie (Hawkins) Worthington. Educated in the public schools of Lovelady, he was graduated in June, 1901, from the Medical Department of Tulane University of Louisiana, New Orleans. He then began his medical career in Creek, where he was located for two years, and practiced in Lincecum, La., and Lovelady before moving to west Texas. He practiced in Marathon for forty years until his death.

A member of the American Medical Association and the

An obituary ordinarily will not be published more than four months after date of death. Cooperation in reporting deaths of physicians and in furnishing appropriate biographical material promptly is solicited.

State Medical Association through Houston County, El Paso County, and Pecos-Jeff Davis-Presidio-Brewster Counties Medical Societies, successively, he was elected to honorary membership in the Association in 1950. Dr. Worthington was a first lieutenant in the medical corps of the Texas Defense Guard. For a number of years he was a local surgeon for the Southern Pacific Lines. In earlier years he was active in the Masonic Lodge, and in August, 1947, was awarded a fifty year pin, being the second member of his lodge to be so honored.

Dr. Worthington married Miss Lydia Hutchings on April 15, 1903, at Lovelady. She survives as well as their two sons, Jim Worthington, Caracas, Venezuela, and Phillip Worthington, McAllen; and a brother, George Clifton Worthington, in Venezuela.

J. B. KNIGHT

Dr. James Bertram Knight, Hamilton, Texas, died October 1, 1950, in a Comanche hospital after receiving injuries in an automobile accident.

Dr. Knight was born November 26, 1891, in Campbell, Hunt County, the son of Robert Paul and Ella (Russell) Knight. Receiving his early education in the public schools of Winnsboro, he was graduated with honors in 1914 from Baylor University College of Medicine, Dallas; in 1922 he took a postgraduate course at Tulane University, New Orleans. After an internship in 1914 at the Emergency Hospital, Dallas, he began his practice in Reinhardt, where he remained for three years. He then served as first lieutenant

DR. JAMES B. KNIGHT

in the U. S. Army Medical Corps, being in charge of a hospital in Deming, N. Mex., during World War I. Dr. Knight after his discharge resumed his medical career as resident physician of Meridian College, Meridian, where he remained for six years. He then practiced in China Springs for eighteen years before moving to Hamilton in 1941, living there until his death. He was a general practitioner.

Dr. Knight was a member of the American Medical Association and the State Medical Association through Bosque County, McLennan County, and Hamilton County Medical Societies, successively. He was a steward in the Methodist Church and a member of the Masonic Order, the American Legion, and Woodmen of the World. He was a former scout master.

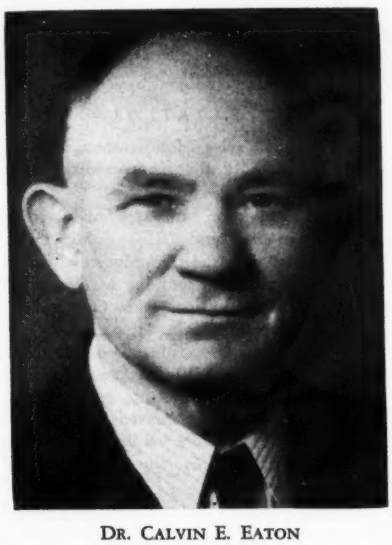
In Waco on January 7, 1938, Dr. Knight married Mrs. Zora (McCrary) Rierson. Surviving are his wife; his two daughters, Mrs. Doris (Knight) Benson, Corpus Christi, and Mrs. Marion (Knight) Mount, Siloam Springs, Ark.; a stepson, M. T. Rierson, Breckenridge; and a brother, R. L. Knight, Imperial Valley, California.

C. E. EATON

Dr. Calvin Eli Eaton, Fort Davis, Texas, died October 26, 1950, in a Brownwood hospital from carcinoma of the prostate gland and generalized metastasis of the bone.

Born January 16, 1881, in Coryell County, Dr. Eaton was the son of George W. and Lucinda Houston Eaton. He attended Brownwood High School, Daniel Baker College, Brownwood, and the Kansas City Veterinary College. For three years he studied at the University of Texas School of Medicine, Galveston, and in June, 1918, was awarded a medical degree from the Medical Department of Texas Christian University, Fort Worth. After internships at City-County Hospital and Harris Hospital, Fort Worth, Dr. Eaton was a house physician and surgeon for the International and Great Northern Railroad Employees Hospital, Palestine. He then practiced in Kilgore until 1921, in Robert Lee from 1921 to 1928, in Alpine, where he was physician for Sul Ross College, from 1928 to 1930, and in Fort Davis from 1930 until his death.

Dr. Eaton was a member of the State Medical Association and the American Medical Association through Pecos-Jeff Davis-Presidio-Brewster Counties Medical Society, being a charter member and president in 1944 of the society. A former county health officer and a member of the local school board for many years, he was county judge-elect at his death. Dr. Eaton was a Methodist and a member of the Masonic Lodge, Karem Temple, Waco; the Eastern Star; and the Mile High Club. He was elected the outstanding citizen of Fort Davis for 1947 by the Mile High Club and presented a physician's bag on behalf of the club at a supper honoring him in 1948.



DR. CALVIN E. EATON

In Waco on December 14, 1918, Dr. Eaton married Miss Pauline Friend, who survives, as do two daughters, Mrs. Grover Jones, Manhattan, Kan., and Mrs. Jack R. Magee, Dallas; seven brothers, D. F. Eaton, Decatur; Pink Eaton, Arlington; H. D. Eaton, Corpus Christi; J. R. Eaton, Hous-

ton; and Lee Eaton, S. J. Eaton, and T. D. Eaton, Brownwood; and two sisters, Mrs. O. W. Ribbles, Lubbock, and Mrs. Cornelia Meadows, El Reno, Okla.

J. F. McKISSICK

Dr. Joseph Franklin McKissick, Arlington, Texas, died in a Fort Worth hospital on October 19, 1950, from chronic nephritis and a stroke.

Born October 1, 1874, in Columbia, Tennessee, Dr. McKissick was the son of Archibald Boone and Paralee Marrow McKissick. Receiving his preliminary education in the public schools in and near Farmersville, he attended Memphis Medical College, Memphis, and in 1905 was graduated from Baylor University College of Medicine, Dallas. After his graduation from medical school, he began practice in Fayburg; he then was located for twelve years in Farmersville

DR. J. F. MCKISSICK

before moving to Arlington. Dr. McKissick practiced in Arlington for forty-two years until his retirement in 1948.

Dr. McKissick was a member of the State Medical Association and the American Medical Association through Tarrant County Medical Society. He belonged to the Baptist Church.

In Farmersville on December 2, 1896, Dr. McKissick married Miss Roxie Baker. She survives as do a son, A. G. McKissick, Fort Worth; two daughters, Mrs. J. H. Jones, Elberton, Ga., and Mrs. Frank Norris, Detroit; one sister, Mrs. Lila Yeaman, West Columbia; and five brothers, George McKissick, Blessing; Hugh McKissick, Pasadena; Luther McKissick, Alvin; and Thomas McKissick and Andy McKissick, Bay City.

A. H. ROBERTSON

Dr. Adolph H. Robertson, Iraan, Texas, died at his home October 17, 1950.

The son of Dr. William Henry and Meta (Tomlin) Robertson, Dr. Robertson was born September 20, 1902, at Canton. He acquired his academic education in the public schools of Frost and Houston and was awarded a bachelor of science degree in 1932 from the University of Texas, Austin; in 1936 he was graduated from the University of Texas School of Medicine, Galveston. After an internship at St. Luke's Hospital, Denver, he practiced in Denver for two years and in Miles, Texas, for two years before moving to Iraan, where he practiced for four and a half years. He owned and operated the Iraan Hospital.

Dr. Robertson was a member of the American Medical Association and the State Medical Association through Pecos-Jeff Davis-Presidio-Brewster Counties Medical Society. He was assistant county health officer in Pecos County. From February, 1941, to February, 1946, Dr. Robertson served in the Army Medical Corps, being attached to the Forty-fifth Infantry Division, 645th Destroyer Battalion, during the North African and Italian campaigns. A member of the Baptist Church, he belonged to the American Legion and the Veterans of Foreign Wars.

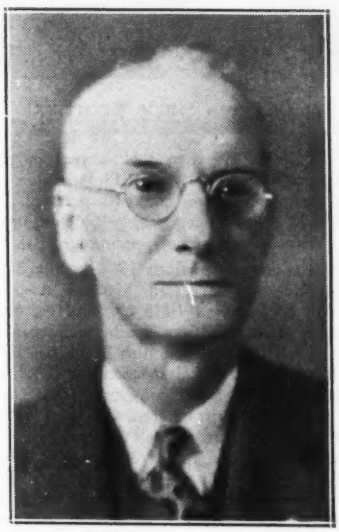
Dr. Robertson married Miss Besse Viola Law on December 30, 1935, in Galveston; she survives as do a daughter, Joan Robertson, Iraan; three brothers, J. K. Robertson and E. H. Robertson, Houston, and H. G. Robertson, Port Neches; and a sister, Mrs. T. B. Farrar, Miles.

H. Y. SWAYZE

Dr. Henry Yandell Swayze, Center Point, Texas, died August 30, 1950, in Center Point from heart failure.

Born in Benton, Mississippi, in 1870, Dr. Swayze was the son of Prentiss and Mary Virginia (O'Riley) Swayze. He attended Centenary College of Louisiana, Shreveport, and was graduated from the Medical Department of Tulane University of Louisiana, New Orleans. Dr. Swayze was located in McCrory, Ark., from 1893 to 1895 and in Benton, Miss., from 1895 to 1918, then moved to Kerrville, practicing there until 1944. He specialized in the treatment of tuberculosis and served as superintendent of the State Tuberculosis Sanitorium, Kerrville, now the McKnight State Sanitorium.

Dr. Swayze was a member of the State Medical Association and the American Medical Association through the



DR. HENRY YANDELL SWAYZE

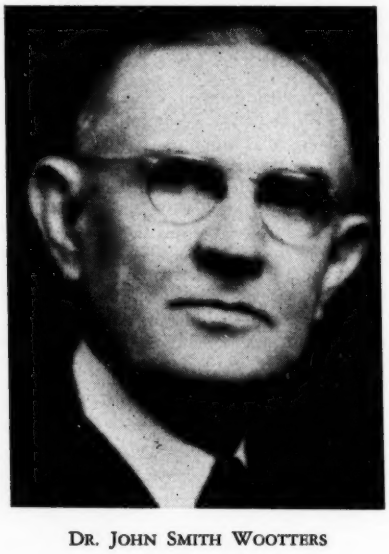
Kerr-Kendall-Gillespie-Bandera Counties Medical Society, formerly having been the society's president. He was a fellow of the American College of Chest Physicians. A Methodist, he was a thirty-second degree Mason and a member of the Rotary Club.

On February 2, 1896, in McCrory, Ark., Dr. Swayze married Miss Jonnie Jelks; she survives as does their son, P. E. Swayze, Center Point.

J. S. WOOTTERS

Dr. John Smith Wootters, Crockett, Texas, the father of Dr. John H. Wootters, Houston, died of cancer October 23, 1950, in a Houston hospital.

The son of Capt. John H. and Berta (Smith) Wootters, Dr. Wootters was born October 3, 1870, at the family plantation on the Trinity River near Crockett. His early education was received in the public schools of Crockett and the University of Texas, Austin, and his medical education in Louisville, Ky., and the Medical Department of Tulane University of Louisiana, New Orleans, from which he was



DR. JOHN SMITH WOOTTERS

graduated in 1891. He then took a year of postgraduate study at Jefferson Medical School, Philadelphia. After practicing one year at Menard and two years at Dailey's, he opened his office at Crockett, where he continued in active practice for fifty-six years, not retiring until this past May.

A member continuously since 1904 of the State Medical Association and the American Medical Association, Dr. Wootters was president in 1935 of the Houston-Anderson-Leon Counties Medical Society. He helped organize the first hospital in Crockett, the Jim Smith Memorial Hospital. Dr. Wootters had served as local surgeon for the International-Great Northern Railway Company and was on the board of deacons of the First Baptist Church, Crockett, for many years. He was city councilman from 1939 to 1947. A charter member of the local Knights of Pythias, Dr. Wootters was Past Worshipful Master in the Blue Lodge; Past High Priest, Past Thrice Illustrious Master, and Past Commander of the Knights Templar in the York Rite Bodies; and a member of El Mina Temple, Galveston. He was a Past Worthy Patron of the Order of the Eastern Star.

On April 21, 1897, in Crockett, Dr. Wootters married Miss Sue Craddock, who survives. Other survivors are two sons, Dr. John H. Wootters, Houston, and Smith B. Wootters, Crockett; three sisters, Mrs. P. R. Denman and Mrs. J. H. Painter, Houston, and Mrs. Willis Higginbotham, Crockett, and two grandchildren.

R. H. CROCKETT

Dr. Roy Hassell Crockett, Tivoli, Texas, died November 5, 1950, in Tivoli.

Dr. Crockett was born in Manor the son of Edward R. and Agnes (Mercer) Crockett August 27, 1882. He was awarded a bachelor of arts degree in 1910 from the University of Texas, Austin, and a medical degree May 31, 1914, from the University of Texas School of Medicine, Galveston. After Dr. Crockett's graduation from medical school, he served an internship at St. Joseph's Infirmary, Houston. He then attended the Graduate School of Medicine of the University of Pennsylvania, Philadelphia, for one year and took postgraduate work at Columbia University and in Memorial Cancer Hospital and Skin and Cancer Hospital, New York, for three months. Dr. Crockett began his medical career in Thorndale, where he practiced until 1922. He was located

DR. R. H. CROCKETT

in San Antonio from 1924 until his retirement in 1947. His specialties were dermatology, syphilology, and radium and x-ray therapy.

A member continuously since 1915 of the State Medical Association and the American Medical Association, Dr. Crockett was a member of Williamson County, Milam County, and Bexar County Medical Societies in successive order. He was elected to honorary membership in the Association in 1949. Dr. Crockett was a member of the Texas Radiological Society, being elected to honorary membership in 1948. He was also a member of the Texas Dermatological Society, the Radiological Society of North America, and the Southern Medical Association. Dr. Crockett was a medical adviser to the Selective Service Board from December, 1940, to March, 1947. He was a member of the staffs of Santa Rosa, Nix, Baptist Memorial, and Robert B. Green Memorial Hospitals, San Antonio. He was chief of the cancer clinic for fifteen years and chief of the skin clinic during six months of each year for twenty-three years at the last-named hospital. He was a member of the Methodist Church and of the Y.M.C.A.

On August 15, 1917, in Galveston, Dr. Crockett married Miss Vernon Richardson, who survives. He is also survived by two daughters, Mrs. J. C. Whittington, Eastland, and Mrs. James F. Crow, Madison, Wis.; his sister, Mrs. W. H. Donathan, Austin; two brothers, M. H. Crockett, Austin, and C. L. Crockett, Houston; and four grandchildren.